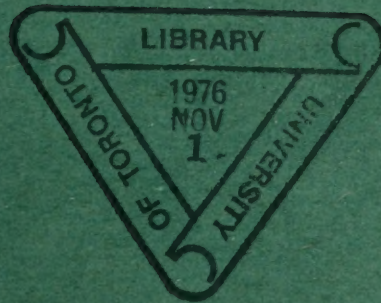


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JUNE, 1912

TRANSACTIONS
OF THE
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Proceedings of a Meeting of the Society on Friday, May 17th, 1912,
SIR HAVELOCK CHARLES, K.C.V.O., *in the Chair.*

A CASE OF SKIN DISEASE OF DOUBTFUL ORIGIN.

SHEWN BY DR. PHINEAS S. ABRAHAM.

The patient I am about to shew has been good enough to come all the way from Wales to shew himself to the Society. It is a case in which I think you will agree the diagnosis is still uncertain. The gentleman is forty-seven years of age; he was born in Scotland, went to reside in South America twenty-seven years ago, and ever since he has been in that country he has been closely connected with cattle. In July last year he noticed three small pimples on the neck, which he supposed at the time followed the bite of some insect. Subsequently, after sixteen or seventeen days, red spots appeared on the body and limbs of the patient. He saw doctors at Buenos Ayres and at some other town in the Argentine, and the affection was at first thought to be some form of erythema, possibly caused by contact with some plant. Subsequently, however, two of the doctors in Buenos Ayres, locally experienced men, came to the conclusion that it was a case of leprosy. The patient then came over here and was seen by Sir PATRICK MANSON—I do not know whether by anybody else in London—who considered that it was a case of leprosy. He then proceeded to Professor UNNA of Hamburg, and was

under his care for two months. Dr. UNNA also looked upon it as leprosy, but could find no bacilli in any of the lesions. Dr. UNNA, however, went away for a holiday, and the young Dr. UNNA tried again, and believed that he had succeeded in finding two or three bacilli in a section taken from the lesion in the neck. The patient came under my care early in February. He at that time presented a number of lesions on his face, a large swelling on the neck, and a great many others of different sizes on the body and limbs. These were raised above the surface of the surrounding skin, brownish red in colour and translucent. As he entered my room, my attention was fixed upon the large lesion on the neck, and I said to him, "Have you lived in a tropical climate?" He said, "Yes; you think I have leprosy." Well, I admitted the fact; at first sight the case did seem to be one of leprosy. At the same time there was something about it that made me have doubts: the lesions were by no means typical, and I had never seen a case exactly like this, and I could find no definite anæsthesia. Some of the swellings were more like the lesions of lupus—multiple lupus; and subsequently, on scraping one of them on the face, I was greatly surprised to find that the tissue broke away just like the soft "apple jelly" tissue of lupus. This made me suspect that it was not a case of leprosy after all, for I have always found the consistence of a leproma to be firmer or more fibrous.

I then, of course, had this tissue that I had scraped away most carefully examined. It was examined at the West London Hospital by Capt. BEAUCHAMP WILLIAMS, who was home at the time, as well as by Dr. BERNSTEIN and myself, and also by Dr. BAYON at the Lister Institute. We all failed to find any of the typical bacilli of leprosy. Dr. BAYON made some serological observations, and found a very faintly positive reaction which might have done for tuberculosis or leprosy. At the same time I tried a Von Pirquet's test, and it showed a faintly positive tubercular reaction. We then came to the conclusion that the disease was probably a form of psorotrichosis, and Dr. FOULERTON, of the Middlesex Hospital, very kindly made further microscopical and cultural examinations. He also came to the distinct conclusion that it was not leprosy, and we were also sure it was not tuberculosis. Dr. FOULERTON, like Capt. BEAUCHAMP WILLIAMS and myself, was, I think, inclined to the diagnosis of psorotrichosis, but his cultures have not proved this. I also sent specimens to Dr. STANLEY GRIFFITH of Cambridge, and he

has made a careful and elaborate series of observations. He was not able to be present here to-night, but he has authorised me to place his observations before the Society. He says:—

“Scrapings from the diseased areas (sent March 4th) were ground fine in a mortar, and made into an emulsion with salt solution. With this emulsion cultures were sown, and two guinea-pigs were inoculated intra-peritoneally. The microscopic examination of a smear preparation of the emulsion did not reveal any acid-fast bacilli.

“The culture tubes (egg medium) produced numerous large coccus colonies, and some small transparent colonies of a bacillus which was not acid-fast.

“The two guinea pigs were killed 28 and 37 days later respectively. In each case the omentum showed a few small soft brownish masses, which were evidently the remains of the inoculum (no acid-fast bacilli found microscopically). The glands and organs of the guinea-pig were perfectly normal. In each of the cases cultures were made from the little masses in the omentum and from various lymphatic glands. These cultures have so far (after more than a month) remained sterile.

“The cord-like structure sent on March 13th was treated in much the same way as the scrapings.”

[Extending from one of the lesions of the thigh this peculiar structure could be felt under the skin, about three and a half inches in length. This, as I suspected, proved to be a swollen lymphatic. I had a section taken of it and an examination made, but I found no tubercle or leprosy bacilli.]

“An emulsion was made of it and inoculated intra-peritoneally into one guinea-pig. A smear preparation of this emulsion showed no tubercle bacilli. The guinea-pig was killed fifty-one days after inoculation, and shewed three little fibrous masses in the omentum. There was nothing abnormal anywhere else in the body of this animal. Cultures were sown with emulsions of the omental bodies (micro.=no acid-fast bacilli), some of the abdominal lymphatic glands and the spleen. All the tubes have so far, nine days after sowing, remained sterile.

“Since in cases of lupus, when similar amounts of original material have been provided, I have not failed to demonstrate tubercle bacilli by the methods employed in this case, it may be concluded that the tissues sent to me did not contain any tubercle bacilli.”

This patient was for some time taking Chaulmoogra oil in large quantities, and I will not say it has not had some effect in diminishing the lesions, but for the last two months, in order to test the effect of the drug, I put him on potassium iodide. At first he commenced by taking 15 grains a day, and he is now taking 90 grains. The lesions have also been locally treated with pure carbolic acid, liniment of iodine, and carbonic acid snow. They are certainly smaller than they were, but they have not diminished as quickly as they ought to have done had it been an ordinary case of psorotrichosis. There is another point which I think is of importance. If it had been leprosy, the taking of so much potassium iodide would probably have had a deleterious effect.

I may add that the patient has always been in the best of health and has had no febrile disturbance, anæsthesia of the extremities, or any affection of the mucous membrane.

The case seemed to me to be of sufficient interest to bring before the Society, and I shall be much obliged for the opinion of the members as regards its diagnosis. Have we here to do with a new disease?

DISCUSSION.

Fleet Surgeon BASSETT-SMITH: A very short time ago I saw a rather similar case, except not so widely spread, in a lady who had returned from South Africa, and here the diagnosis between leprosy, syphilis, and tuberculosis was just the same. The spots increased in number and continued to spread over the forehead, which gave her the most trouble. They were present over the hands, knees and other parts. Syphilis was excluded. One of the patches, slightly raised like these and brown in colour, was cut out and sections were made. Personally I think it was some new form of xanthoma. I sent a specimen then to Dr. SEQUEIRA, in London, and he, with Dr. TURNBULL, of the London Hospital, both came to a similar conclusion, and up to the present the diagnosis of that case is xanthoma, and this case seems to me to be similar.

PRIMITIVE TRIBES AND TUBERCULOSIS.

By MAJOR S. L. CUMMINS, R.A.M.C.

Some years ago, when engaged in preparing a paper on Tuberculosis in the Egyptian Army, I was struck by the fact that the incidence of this was much higher amongst the Sudanese than the Egyptian troops. The paper in question duly appeared in the Service Number of *The British Journal of Tuberculosis*,¹ published in January, 1908, but I may quote some figures from it to illustrate the point in question.

For the five years from 1902 to 1906 inclusive the average annual incidence amongst the Sudanese was 3·7 per 1,000, whilst amongst the Egyptians it was only 1·5 per 1,000.

This difference was significant, but the contrast was much more intense in Cairo, where the climate is colder and where town conditions obtain. During twelve months in 1906-7, fifteen acute cases of tubercle of the lungs came under my notice. Of these, ten were either transfers from other stations or admissions from the Egyptian garrison of about 3,000. No less than five cases, or one third of the total, were contributed by a small detachment of sixty or seventy Sudanese Artificers stationed at the Citadel for instruction.

An Egyptian medical officer, to whom I mentioned these points, told me that this great liability of the Sudanese to tuberculosis was well known in Egypt, so much so, that in former times, Shilluk and Dinka slaves were the cheapest in the market, as it was assumed that a large number would contract the disease and die.

VON BECKER² has called attention to the small incidence of tuberculosis amongst Egyptians in Egypt and contrasted it with the intense susceptibility to the disease shown by Berberines, and still more by Sudanese, when the latter are under Egyptian conditions of life. Still, tuberculosis exists and has long existed amongst the people of Egypt. ELLIOT-SMITH³ has found tubercular lesions in the bones of ancient Egyptians, and tuberculosis is common amongst cattle in Egypt. On the other hand, *the Sudanese soldier's great liability to the disease is accompanied by an almost complete absence of it amongst the primitive Sudanese tribes in their natural surroundings, and by a corresponding absence of tubercle in the herds of the Sudanese cattle-*

owning tribes. In my own experience, as well as in that of several brother officers who had served in the Bahr-el-Ghazal, no cases of tuberculosis had been seen in natives except in the vicinity of Government posts, where contact with the troops was frequent. At the time of writing the paper which has been mentioned, there remained some doubt as to whether the observations mentioned dealt with a sufficient number of wild tribesmen to be of significance. But more recent information leaves no room for doubt on this point. Capt. R. G. ANDERSON, R.A.M.C., to whom I wrote for information, states:

"Amongst the Nyam-Nyam and Gours I did not meet with a single even suspicious case."

The evidence of Capt. C. M. DREW, R.A.M.C., on this subject may, I think, be taken as conclusive. While on sleeping sickness work in the Lado he never saw a case of tuberculosis amongst the tribes (Kakwa, Fadjelu, Makaraka, Avukayja, Bari and Mundu) coming under his observation, although large numbers were available for examination. For instance, he palpated the glands of just short of 10,000 people without finding any cases of tubercular adenitis or other manifestations of the disease.

To reiterate, then, the Egyptian soldier proceeding to the Northern Sudan from Egypt—a country where tuberculosis is apparently common—has a decidedly low tubercular case incidence, whilst the Sudanese soldier, recruited from the southern tribes amongst whom tuberculosis is rare or absent, shows under military conditions a case incidence which is decidedly high.

This apparent paradox is by no means an isolated instance, but appears to be the expression of a great rule in the epidemiology of tuberculosis. I will quote a few of the many recorded examples.

MACVICAR,⁴ writing on tuberculosis in South Africa, calls attention to the high incidence of the disease amongst coloured people when in contact with Europeans, and mentions that "in the interior, especially in the Transvaal and on the Upper Zambesi, tuberculosis is almost unknown." At the same time, the tubercle death-rate amongst whites compares favourably with that in European countries, being 1·4 per 1,000 in thirty-five towns of Cape Colony, 1 per 1,000 in Natal, 1·37 per 1,000 in Orange River Colony, as compared with 1·62 per 1,000 in England and Wales during 1902.

The average death-rate of coloured people in six towns of the western province of Cape Colony was over 8·6 per 1,000. So that here there is an exact analogy to the Sudanese paradox.

The European, coming to South Africa from a country where tuberculosis is common, has a low incidence from that disease, while the native, coming from the interior where tuberculosis is rare or absent, displays, when he mingles with the European, an intense liability to tuberculosis.

The rarity or absence of tuberculosis amongst natives in Central and German East Africa has been noted by R. KOCH⁵ and by German colonial medical officers.

RÖMER⁶ has recorded similar observations from the Pampas of the Argentine, where tuberculosis is rare amongst the inhabitants. They suffer severely from it when they settle in the towns, much more severely than people of the same race permanently domiciled in the cities of Buenos Ayres.

The North American Indian affords another terrible instance to the same effect. Thus, writing on KLEB's work on "Tuberculosis," Dr. THOMAS D. COLEMAN says: "It is generally conceded that pulmonary tuberculosis was unknown in the Indian in his barbaric state—at least that it was a disease of rare occurrence." Then, giving examples of the present incidence of the disease amongst these people:—"In 1896 there were altogether 4,893 Oglalas, of whom 741 were tuberculous, and of these 124 died in one year. That is, 148·7 per 1,000 were tuberculous and the annual death-rate was 24·88 per 1,000." Dr. COLEMAN gives other examples as striking and as terrible as this one.

America furnishes another illustration in the case of the negroes. These Africans, under the old slave-régime, lived on the plantations under much the same conditions as in their native homes, with the difference that, being valuable property, they were carefully looked after by their owners. COLEMAN says the medical profession in the South is unanimous in the opinion that in the negro's condition of slavery tuberculosis was practically unknown.

In 1900, however, the state of things had become very different. "In the cities of the South, with a negro population ranging from 27 per cent. to 56 per cent., the death-rate of the negroes from consumption is two or three times that of the whites; in Boston, 7·4 per 1,000 in negroes and 2·3 per 1,000 in whites; in New York, 5·2 per 1,000 in negroes and 2·3 per 1,000 in whites; and so on."

Major F. SMITH, D.S.O.,⁷ has called attention to a similar high incidence amongst the men of the West Indian Regiments, both in the West Indies and when stationed at Sierra Leone, as compared with the incidence amongst European troops in the same stations and also as compared to that amongst the West African Regiment recruited from the natives of the West Coast. The latter comparison might be misleading if it were not borne in mind that the high mortality amongst West Indian troops stationed at Sierra Leone is not the expression of their response to tuberculosis contact on the West Coast, but of their previous contact with it under West Indian conditions; an exposure evidently much greater than that to which the recently raised West African troops have been subjected.

The last instance that I shall mention is that of the Tartar Kalmuks, recently investigated by METCHNIKOFF, BURNET and TARASSEVITCH.⁸ This nomadic tribe, fifty years ago, was practically free from tuberculosis, but, under the influence of gradually increasing contact with the Russians, it is becoming more and more infected with the disease. Still, however, cases are rare in the central parts of the area inhabited by the tribe, and still the same phenomenon as has been noted above in the Sudan is displayed by the Kalmuks at the periphery of their country, where foreign contact is the rule—a very great susceptibility to the disease. So much is this the case, that students in the colleges at Astrakhan very seldom complete their studies, being obliged to cut them short through infection with tuberculosis, and the shutting up of several schools and their transfer to the interior of the country is now contemplated. Other instances to the same effect could be given, but it is unnecessary to labour the point, which is made sufficiently clear by those already quoted. There is then much reason to believe, (1) *That man, living under primitive conditions, is practically free from tuberculosis,* and (2) *That man, when brought from a state of primitive existence and placed in contact with civilisation, shews a very much greater liability to tuberculosis than does civilised man himself.*

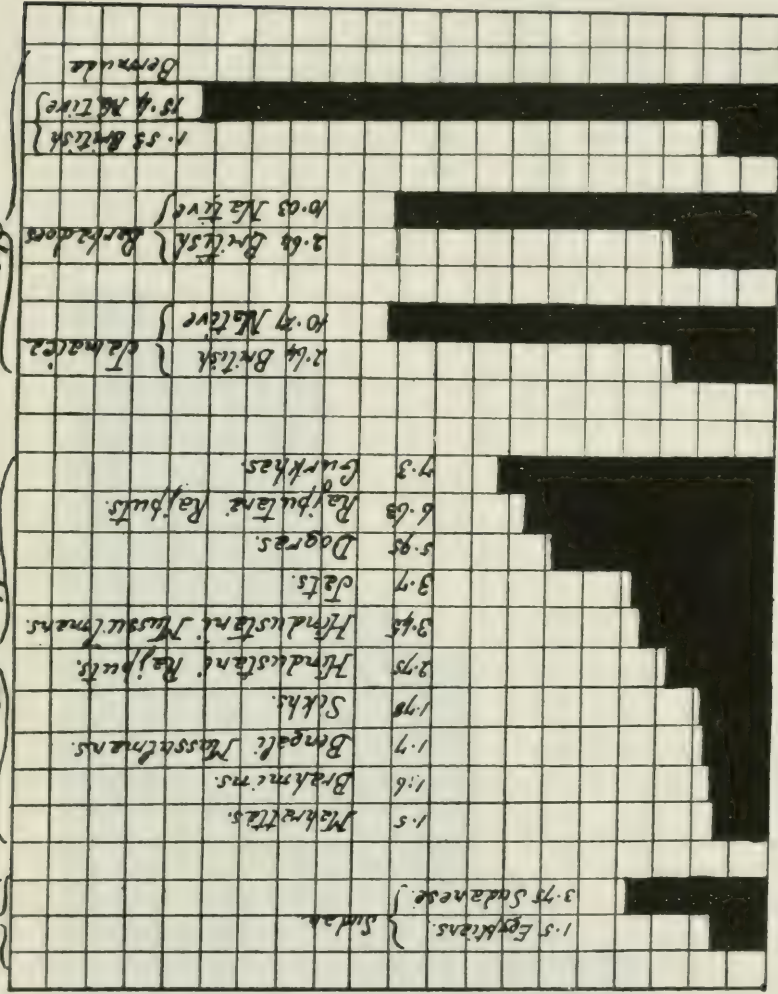
The question then arises whether the second of these propositions is a result of the first; *whether, in short, primitive man owes his liability to the disease to the fact that, having no previous contact with the tubercle bacillus, he is, as it were, virgin soil for that organism.*

This is the explanation that suggested itself to me on investigating

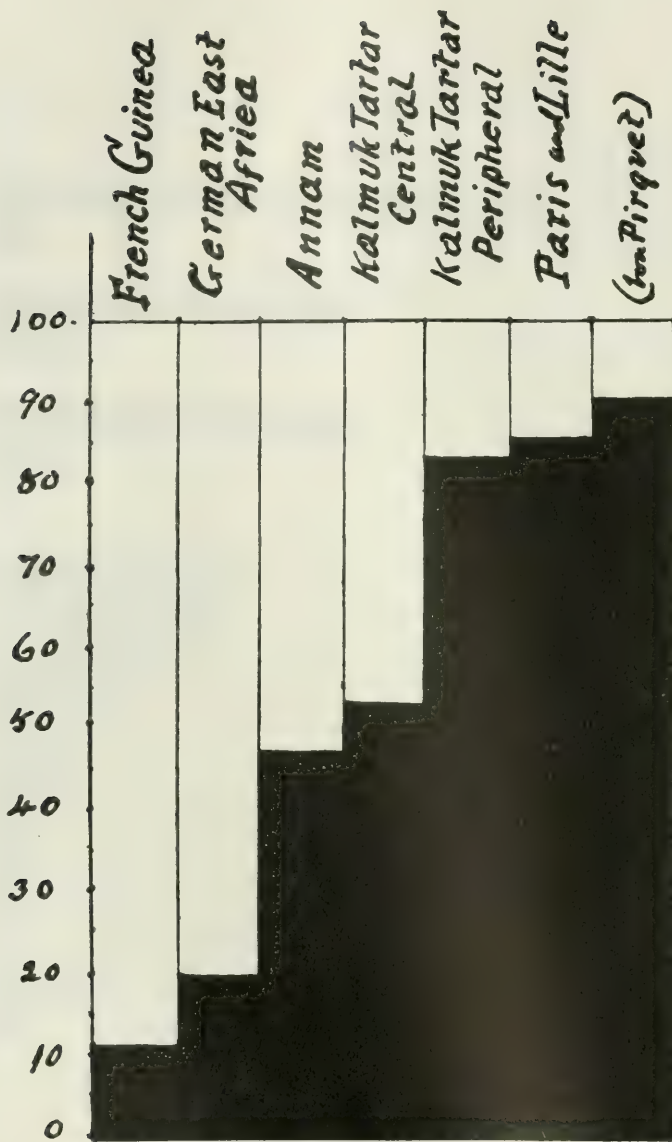
Egyptian Army.
1902-1906.

Indian Army.
1885-1899

West Indies.
1898



Tuberculosis incidence per mille in different races under military conditions.



Comparative percentages of positive cutaneous tuberculin reactions in primitive and civilized peoples.

the incidence of tuberculosis in the Egyptian Army. I have since learnt that much more competent observers, such as RÖMER, METCHNIKOFF and BURNET, having similar facts to explain, have come to the same conclusion. But cannot the increased incidence be explained in any other way?

Is it not natural to suspect that these wild people on being placed under the clothed, housed and overcrowded conditions of civilisation should rapidly deteriorate? Yes, but why specially through tuberculosis? And it is open to question whether the sanitary surroundings of primitive man are ideal. It must not be supposed that the savage insists on over 600 cubic feet of air-space in his hut or tepee. The Dinka hut has only one opening, a door, entered by creeping on the hands and knees!

The building measures about twelve feet by twelve feet, and it is usual to light a fire in it at night as a precaution against mosquitoes. I should not like to say how many people occupy it, but limitation of families has not yet become a problem in the Bahr-el-Ghazal.

Dr. JAMES R. WALKER,⁹ speaking of the North American Indian, says, "They were filthy both when they lived in houses and when they lived in tepees." Of the Kalmuks, who fifty years ago were almost free from tuberculosis, and even to-day have but few cases in the central districts of their country, METCHNIKOFF and his collaborators remark that "All the diseases that are transmitted by contact, and flourish especially amongst unclean people, are widely spread among the Kalmuks."

It is not to be denied, however, that uncivilised man usually makes a change for the worse in regard to his surroundings when he comes to or is reached by civilisation, but while this might explain his being *as* liable to tuberculosis as the civilised people with whom he mingled, it cannot account for his being much more so.

So far, then, the facts are in favour of the virgin soil theory. But this theory commits us to an inference. If the Sudanese, for instance, is liable to tubercle because he is virgin soil, then the healthy Egyptian or European must present to the tubercle bacillus a soil that is other than virgin.

It is not enough to say that there has been contact with tubercle. The contact must have involved a struggle, the struggle must have ended in victory—temporary or permanent—for the human organism over the

tubercle bacillus. And this victory can only be expressed in terms of a relative immunity to the bacillus. Is there, then, any evidence that such a victory has been obtained by the average healthy civilised man?

Perhaps the most striking evidence is that of NÄGELI¹⁰ and of BURGHARDT,¹¹ dealing with the post-mortem findings of large numbers of persons dying from all causes.

Both these observers have shewn that the percentage of persons with tubercular lesions gradually rises in different age-groups, attaining to 92 per cent. (BURGHARDT) or 96 per cent. (NÄGELI), between the ages of twenty and ninety years. In other words, a large number of persons in civilised communities have contracted and overcome focal infections.

But it may be objected that these were hospital cases, and that results founded on such material cannot be applied to the general population.

Fortunately there is evidence available upon this point also. The Von Pirquet cutaneous tuberculin test, and the ophthalmo-reaction of CALMETTE, provide a specific method of ascertaining whether persons have or have not tubercular deposits in their tissues. These tests, applied to healthy people, demonstrate that a large proportion of adults in European countries react to tuberculin. Thus MANTOUX¹² and CALMETTE GRYZEZ and LETULLE¹³ have shewn that from 80 to 90 per cent. of adults in Paris and in the neighbourhood of Lille give a positive reaction to the cutaneous test.

On one occasion, when asked to apply the Von Pirquet test in the diagnosis of cases at Netley Hospital, I selected ten apparently non-tubercular soldiers as a "control." They were carefully examined to exclude clinical manifestations, and their medical history sheets shewed no suspicious entries during their service. Nine out of the ten gave a decided cutaneous reaction.

So much for adults, but the picture is quite different when it comes to children. VON PIRQUET'S¹⁴ beautiful observations on this point make it clear that while hardly any healthy infants respond during the first two years of life, the number that react to the test rapidly rises with increasing age, until at between thirteen and fourteen years, the percentage amounts to the same figure as that for adults—80 to 90 per cent. It is clear, then, that the adult European is in no sense of the word virgin soil for the tubercle bacillus.

But what of primitive man? Has he been subjected to such tests?

The information on this point requires to be supplemented by much additional research, but what work has been done all points to the conclusion that primitive adults only react in small numbers.

For instance, WAGON¹⁵ applied both the cutaneous and the ophthalmic reactions to one hundred natives at Kindia in French Guinea, with the result that he obtained only twelve positive results. Of the twelve persons responding to the test, eleven had left their country and been much in contact with Europeans. PEIPER¹⁶ has obtained similar results in German East Africa. It must be remembered, however, that the skin of the negro may possibly to some extent obscure the response to the Von Pirquet test. This objection cannot be urged against the very important researches of METCHNIKOFF, BURNET and TARASSEVITCH amongst the Kalmuk Tartars.

The Kalmuks at the periphery are tending to lose their nomadic habits and becoming associated with Russians to an increasing extent. The number of positive reactions in adults at the periphery was 82 per cent., or about the same as amongst civilised Europeans, while in the more central parts of the country, away from much foreign contact, the proportion in adults was only 50 per cent.

BERNARD, KOUN and MESLIN¹⁷ have made an important contribution to the subject in a recent paper on "The Epidemiology of Tuberculosis in Annam." The people of this country have been in contact with Europeans for upwards of fifty years, and the observations were apparently carried out on persons living under urban conditions, but here too the proportion of persons giving a positive response to the tuberculin test was decidedly low, being intermediate between that of Africans cited by WAGON and PEIPER on the one hand and of the Tartar Kalmuks examined by METCHNIKOFF and his co-workers.

It will be seen that there is reason to believe that while the healthy adult who has grown up in civilised surroundings has, in ninety or more per cent. of cases, a demonstrable but isolated and harmless tubercular focus somewhere in his body, the African, under the natural conditions of his own country, is demonstrably free from such foci in 80 or 90 per cent. of cases; while races, like the Annamese and Kalmuk, subjected in increasing degree to contact with civilisation, occupy an intermediate position.

Now, since a latent tubercular focus must have been rendered latent by the acquisition of sufficient immunity to prevent its spread, and must usually be kept latent by the acquired anti-bacterial properties of the body-fluids, the 96 per cent. of latent tubercular lesions found by NAGELI means that 96 per cent. of the people whom he examined possessed or had possessed a certain degree of immunity to tubercle bacilli, and the 80 or 90 per cent. of Africans that failed to respond to WAGON'S tuberculin tests were demonstrably wanting in such an immunity.

Here, then, are the conclusions to which we are driven if we accept the virgin soil theory :—

1. *That primitive tribes are highly susceptible to tuberculosis, because, in the absence of the tubercle bacillus, they have never been obliged to protect themselves against that organism.*

2. *That civilised peoples are highly protected against tuberculosis because they have, in contact with the tubercle bacillus, elaborated protective substances against that organism.*

The first proposition lays upon us a duty and the second opens to us the hope of fulfilling it with success.

The duty is one that lies in a very special degree upon the Fellows of this Society, since our work lies so largely amongst the primitive tribes of the Colonies and Dependencies of this great Empire. If it is true that the African, for instance, is specially liable to tuberculosis, then it may spread rapidly in the Sudan, in Uganda, in East Africa, when once it is introduced. Our efforts should be directed to keeping it out, by stringently forbidding the country to invalids seeking a climate suitable for early tuberculosis, by invaliding every official who contracts or develops tubercular disease, and above all by detecting and isolating early cases amongst the natives.

Again, in the case where the natives leave their country to take service in our regiments or to seek employment in our industries, we should protect them as far as possible from contact with tuberculosis by a rigid inspection and selection of their officers, instructors and overseers, by careful attention to their housing and general sanitation, and by strict regulations that invalids passed out of the service for tubercular disease are not sent back to their native villages.

The problem of getting rid of tuberculosis where it has once gained a secure footing is perhaps too difficult for solution, but it may not be

beyond the power of foresight and organisation to keep it from gaining a footing in countries where it has not yet appeared. Even a very susceptible person cannot get tuberculosis unless he comes in contact with the tubercle bacillus, and much may be accomplished on lines of preventing such contact especially where there is not great density of population.

To show how successful such methods may be, I may quote the following instances of an analogous kind. The great susceptibility of monkeys to tubercular disease is well known. In their wild state, these animals appear not to suffer at all, but their mortality in captivity is notorious. Captain S. FLOWER, Director of the Zoological Gardens at Cairo, tells me that, whereas formerly he lost numbers of monkeys from tuberculosis, he has only had one death from the disease in the last three years. The deaths ceased when he moved the cages back so as to be out of reach of visitors, who are now unable to handle or feed the animals, and who can no longer spit into the cages, a favourite amusement of the human being when he interests himself in his Simian relatives.

Dr. PLIMMER, who most kindly provided me with information on this subject, has succeeded in greatly reducing the death-rate from tuberculosis amongst the large number of monkeys in the Zoo in Regent's Park by a search for and rejection of infected animals and a thorough cleaning up of the cages; after which a rigid rejection of all arrivals that showed the least trace of tuberculosis prevented the introduction of fresh cases.

By this means the annual percentage of deaths amongst primates from tuberculosis has been reduced from 17·2 per cent. in 1908 to 3·7 per cent., 3·9 per cent., and 3·2 per cent. in the last three years.

For the last twelve years, there has been no death from tuberculosis amongst the anthropoids at the Zoo, though the susceptibility of these animals to the disease has been proved, for chimpanzees at least, by the work of the Royal Commission on tuberculosis, but during all that time these animals, unlike the lower primates, have been kept behind glass, and have not been exposed to the direct attentions from the public that are enjoyed by the monkeys.

There is much, then, to be done in the direction of protecting susceptible races from contact with the bacillus; but it must be admitted that the task will be more and more difficult as civilisation overflows its confines on the one hand, and as native populations increase under the

conditions of peace and material prosperity that our administration brings to them on the other. The more difficult the problem, the more skill and knowledge do we require to bring to it; and knowledge is only gained by *research*.

The questions still unsettled as regards tuberculosis are many, and a large number of them should appeal in especial degree to the Fellows of this Society. To begin with, many of you will, no doubt, regard what I have called the virgin soil theory as in need of further evidence before it can be accepted. I hope I may at least have shown that it demands being either accepted and acted upon or refuted and put aside. Most of the evidence bearing upon the point comes from France, Germany or America. We furnish but little. It is not that we are in need of material to work on. India alone presents vast problems. Major C. A. JOHNSON, I.M.S.,¹⁸ has called attention to the very uneven distribution of tuberculosis in the Native Army amongst regiments of different race. The Ghurkas for instance have an incidence of 7·3 per 1,000, the Dogras, 5·9; while the Sikhs, Panjabi Mussulmans, Brahmins and Maharattas vary from 1·8 to 1·5 per 1,000.

Do the Ghurkas and Dogras suffer to this extent in the villages of their native hills?

Do the recruits joining the army from Nepal react to the cutaneous tuberculin test in as high a proportion as those recruited in the Punjab?

These questions would well repay organised research. But, above all, research is needed in the direction of the artificial protection of susceptible people by means of inoculation. If the inhabitants of countries where tuberculosis is widespread tend to acquire an immunity against the disease, the prospect of reproducing this phenomenon by measures under accurate control cannot be quite hopeless. Nature is rough and ready in her methods, and her process of immunising a race is fraught with many an overdose and many a "negative phase." Perhaps it is presumptuous, but may we not venture to hope for better results in working scientifically along the lines that she has indicated? Work is being done in this direction already. VON BEHRING has attempted to immunise calves, and promising results have been obtained in the case of man himself. The attempt to immunise susceptible persons coming from primitive tribes to our armies or to our mines might lead to results of far wider importance than any question of purely tropical medicine.

But while efforts at artificial immunisation are still in the experimental stage and demand research rather than active application, we have it in our power even now to assist nature in her vast campaign against tuberculosis. She is handicapped by the artificial conditions of our civilisation. She aims at an immunising dose. Overcrowding and overwork make it a lethal one. Once we accept the point of view that primitive peoples are not the victims of an inevitable and invincible racial susceptibility, but are merely on a par with the children of our own race, we are in a better position to influence those responsible for administration on behalf of the tribes that they are attempting to civilise and improve. In conclusion, I wish to own my indebtedness to Dr. ETIENNE BURNET, Sir WILLIAM LEISHMAN and Dr. BALFOUR for references to the literature bearing on this subject, and to Captain FLOWER, Dr. PLIMMER, Captain ANDERSON and Captain DREW for invaluable information and help in compiling this paper.

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TWO CASES OF TUBERCULOSIS IN AUSTRALIAN
ABORIGINES WITH NODULAR DEPOSITS
IN THE VISCERA.*

BY J. BURTON CLELAND, M.D., CH.M. (SYDNEY).

Thanks to the courtesy of Dr. W. E. BLACKALL, of Perth, Western Australia, and of Dr. C. REISSMANN, of Adelaide, materials from two very interesting cases of tuberculosis in Australian natives have been made available to me for examination.

The Australian aboriginal is fast disappearing. In the settled portions of the south of the continent pure-blooded individuals are rarely seen, and in some of the States are already almost extinct. It seems of value, therefore, to record every occurrence of disease amongst them, and still more so when the manifestations of such disease seem to differ from those usually found in Europeans. It is with this object in view that the two following cases of tuberculosis in Australian natives are recorded, prefaced with a short note on previous references to this disease amongst the aboriginals.

The idea seems wide-spread that tuberculosis has been one of the causes at work in aiding the rapid disappearance of the native race, but actual references to its relative frequency seem few, and published cases, dealing with its clinical or pathological aspects almost unknown. As valuable records of the disease amongst them, however, the following may be quoted. Professor STIRLING[†] of Adelaide, says: "The extreme susceptibility of the Australian race to tuberculosis, when brought into contact with civilisation, has frequently been observed, but it is difficult to say whether a similar tendency exists in their natural condition." After referring to climatic influences and rapid changes of temperature as being likely to cause a heavy percentage of the total mortality, Dr. STIRLING says: "There was no obvious evidence that such was the case, and there is a good deal to be said in favour of the view that the great tendency to lung complaints, observed in the natives who have come under the influence of civilisation, arises from the fact that they

* This Paper was presented to the Meeting on May 17th, 1912, but not read.

† Dr. E. C. STIRLING, C.M.G., F.R.S., *Report on the Work of the Horn Scientific Expedition to Central Australia, 1896. Anthropology*, Vol. iv., p. 127.

alternate periods of being well clad, sheltered and fed, with those in which they revert to the conditions of complete or semi-nudity, exposure and hard living, peculiar to their natural or wild life.

"My colleague, Professor WATSON, informs me that in the natives who have died in the hospital the invariable seat of commencing tuberculosis is the lymphatic glands, which condition runs on to general disseminated tubercle. In no case, out of a considerable number of autopsies, has he seen excavation of the lungs.

"During a recent investigation of the records of the Adelaide Hospital the remarkable fact appeared that no aboriginal admitted as an inpatient during the last eleven years has died in the institution from any other cause than tuberculosis, hydatid disease, or accident."

The Rev. DONALD MACKILLOP, S.J.,* in speaking of the aboriginal tribes of the Daly River in North Australia, says: "As a rule they die by murder, of consumption, or of old age."

The two cases, the special subject of this paper, are characterised by the organs containing large, somewhat firm, white nodules, suggestive in the one case of malignant deposits, in the other of those of lymphadenoma. In both cases, however, the nodules proved on microscopic examination to consist of necrosed tissue becoming caseous, and acid-fast bacilli were detected in each. Giant-cells were few in number and, as a rule, small in size. In both, miliary nodules were scattered over the peritoneum. In Dr. REISSMANN'S case a single small cavity was present in the lungs, whilst in Dr. BLACKALL'S case only nodules, and no vomicae or ordinary signs of phthisis. In a general way, therefore, these two cases support Professor WATSON'S experience as regards the absence, or at least rarity, of excavation of the lungs.

CASE 1. *Beria-Beria* (*Berra-Berra*, "Sugar").—For the excellent post-mortem notes of this case and for sections of the issues, I am indebted to Dr. W. E. BLACKALL, of Cottesloe, West Australia. As the post-mortem account indicates, the diagnosis made on the table was that of malignant disease, and it was only when the microscopical examination of the tissues came to be made that the true nature of the case was

* *Trans. and Proc. Roy. Soc. of S.A.*, Vol. xvii., pt. ii., 1892-3, pp. 254-258.

discovered. Obviously, therefore, the condition macroscopically did not resemble typical cases of tuberculosis in Europeans.

The patient was admitted to Freemantle Lunatic Asylum on May 16th, 1894; he died on March 25th, 1905, 11.30 p.m. Diagnosis of mental condition—mania. Post-mortem on March 26th, 11 a.m.

Summary of results.—Yellow fluid and coagulated lymph in both pleural cavities. Pneumonia was present and rounded yellowish nodules were scattered in the lungs. Gangrenous glands along the trachea. Thickened visceral pericardium. Visceral peritoneum studded with small nodules. Small rounded nodules in the spleen and kidneys. A tough mass near the pylorus and the head of the pancreas.

The detailed description of the post-mortem is as follows:—

Lungs.—Both pleural cavities were full of clear yellow serum, with masses of clear yellow coagulated lymph. Slight and very recent adhesions in front. The parietal pleura was thickened with a deposit of yellow opaque lymph. The visceral pleura was greatly thickened, being tough and fibrous: the left lung was very small and contracted right away from the chest wall. It was solid and pneumonic, and numerous round yellow nodules were scattered throughout, never larger than a small pea, not caseous or calcified. There were no vomicae or signs of phthisis. The right lung was similar to the left except that it was not quite so pneumonic, a little air being able to be squeezed out. The glands at the bifurcation of the trachea were enlarged and gangrenous, giving off a most offensive odour. The chain of gangrenous glands extended up the trachea almost to the cricoid cartilage. Above this the glands were enlarged and of a maroon colour. On the left side they were enlarged, hard, and looked typically cancerous.

Heart.—The pericardium was greatly thickened and, on opening it, the heart was enlarged and not adherent in front to the pericardium. The heart was covered with a rough, red, soft deposit, which quite obscured the auriculo-ventricular groove, and below the heart was loosely adherent to the pericardium by this blood-stained lymph. On cutting into the heart after removal it was found that the visceral pericardium was much thickened, white and fibrous with the soft red lymph outside. The muscle substance was pale and diminished in thickness, the chambers were dilated, but the valves normal. The aorta was free from atheroma. The heart chambers contained white thrombi.

Liver.—Small. Capsule thickened and firmly adherent to the diaphragm. On section, fatty with small cancer-like areas deposited on the peritoneal surface.

Stomach.—The stomach wall was not invaded nor the pylorus thickened. A few scattered deposits were found on its peritoneal surface.

Pancreas.—Appeared normal, but the head was invaded by a tough mass situated in the region of the pylorus.

Peritoneum.—The parietal peritoneum was little invaded, but the visceral part was studded throughout, especially in the mesentery and over the true pelvis.

Kidneys.—Invaded with small round masses. They were pale and large.

Spleen.—Large, and its substance likewise invaded. Not many deposits on it superficially.

Testicles, bones, etc.—Seemed normal. A thick mass was found in the omentum.

Brain.—Shewed nothing abnormal macroscopically, except dilated lateral ventricles filled with cerebro-spinal fluid.

EXAMINATION OF MICROSCOPICAL SECTIONS.

Thickened pleura and detached lung.—A few small giant-cells and some areas of necrosis were present. In these situations were many plasma cells and round cells. The lung alveoli contained a few catarrhal cells. There were very numerous acid-fast bacilli in places, not where the necrosis was, but near it. In the lung tissue which was infiltrated was a fair number of plasma and round cells. Giant-cells were not seen near the bacilli.

Pericardium and attached heart muscle.—There was an infiltration with plasma and round cells extending in between the muscle. No typical giant-cells were seen. Patches of acid-fast bacilli were noticed in two cases, aggregated into masses of ten or so, apparently in cells.

Kidney.—Sections shewed considerable disorganisation, only the remains of the tubules appearing here and there, and no Malpighian capsules. The remains were separated by plasma and round cells and blood corpuscles. A few scattered areas occurred shewing occasionally a small amount of caseation in the centre, and not frequently small imperfect giant-cells. A number of collections of gold and yellow

pigment, especially around small vessels, appeared in some of the cells. Tubercle bacilli were not detected.

Spleen.—The spleen shewed typical tubercles with large giant-cells and small areas of caseation. Tubercle bacilli themselves were not detected.

Gland in the neck.—The tissue stained very poorly, and consisted apparently of degenerated cells. Amongst these tubercle bacilli were not recognised.

CASE 2. The specimens from this case were shewn by Dr. REISSMANN at a meeting of the South Australian Branch of the British Medical Association, on the 30th September, 1909. They consisted of the lung and spleen, and Dr. REISSMANN remarked on the resemblance which these organs bore to the condition found in Hodgkin's disease (*Australian Medical Gazette*, Nov. 20th, 1909, p. 609). Dr. REISSMANN kindly forwarded the specimens to me, and from these the following description was drawn up:—

The spleen is sectioned in two. Near the hilum is a folded hydatid cyst, about $2\frac{3}{4}$ ins. in longest diameter. Between it and the spleen substance proper, is a layer of yellowish caseous tissue with occasional slight hæmorrhages, $\frac{3}{8}$ in. thick, but separated by spaces from the spleen tissue and the cyst. A prolongation of this tissue occurs for $1\frac{3}{4}$ ins. beyond one end of the spleen as a thick yellowish area $\frac{3}{4}$ in. in diameter, with extravasated areas of blood. This abuts directly on the spleen capsule, and, on the outside of the spleen, interposes as a narrow band between the spleen capsule and the spleen tissue for a short distance, whilst for the rest of the surface the splenic capsule is markedly thickened.

Shewing up through the dark red splenic tissue are numerous scattered yellowish-white areas with frayed edges, round or oblong, the largest being $\frac{5}{8}$ in. long, the smallest pin-point.

Section of lung.—Near the periphery is a cavity $1\frac{1}{2}$ in. by $\frac{5}{8}$ in., with a ragged wall in which some yellowish areas are visible. The wall of this cavity is thin and reddish-yellow. Throughout the rest of the lung substance are very numerous irregular yellowish-white areas, mostly under $\frac{1}{4}$ in. diameter, usually showing one or more small openings (bronchioles?). A small bronchiole in longitudinal section shows its distal part and branches occupied by yellowish growth. The intervening

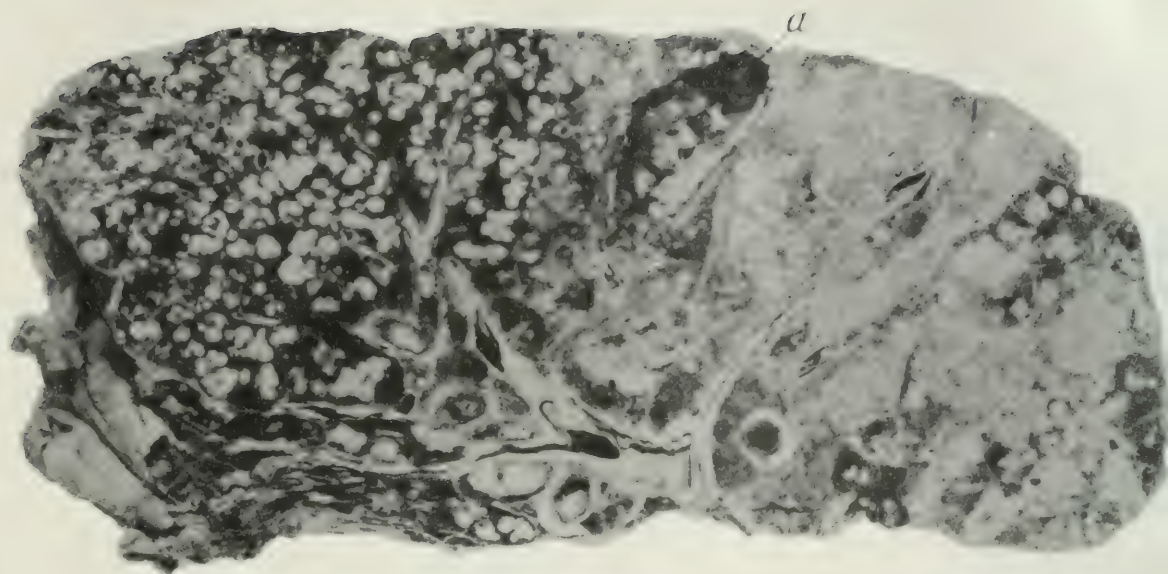


Fig. 1 — Lung.
Dr REISSMANN'S Case. a—Small Cavity.

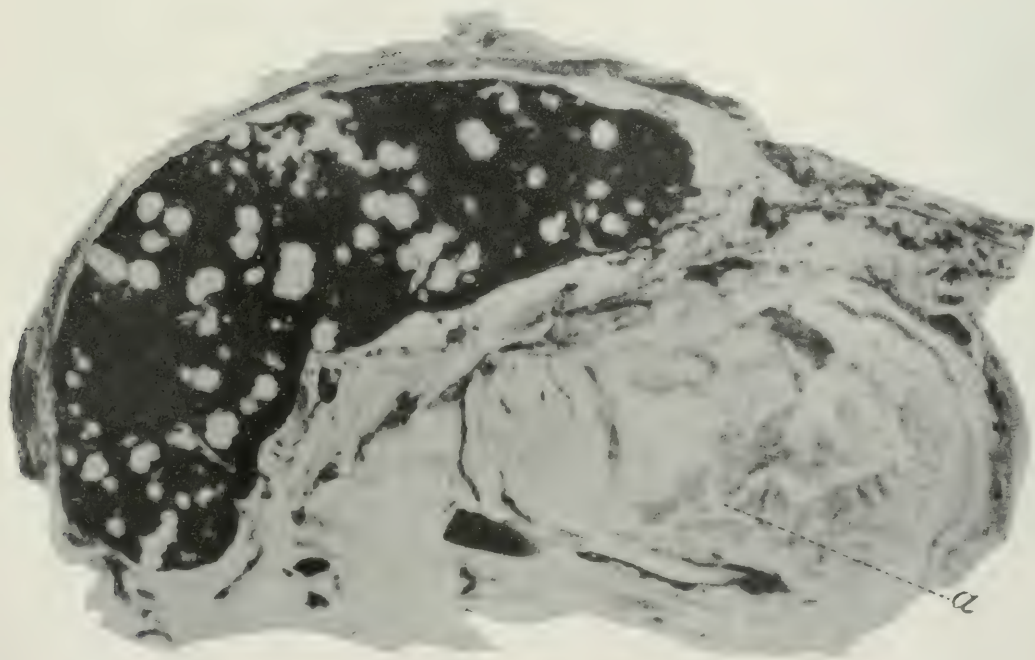


Fig. 2.—Spleen.
a—Hydatid Cyst.

pulmonary tissue is dark red and consolidated looking. In another part of the section the yellowish-white areas are much more extensive, having run together into large masses and the intervening pulmonary tissue is becoming greyish-white.

The *microscopical examination* of these tissues shews the following :—

Spleen.—A section through the splenic pulp containing several small caseous areas shews varying sized areas undergoing caseation, which stain poorly and consist of degenerated cells and pigmented nuclei. No giant-cells were seen. The caseous areas seem scattered around vessels, more on one side than another. A few tubercle bacilli only were seen. In the thickened capsule were similar caseous areas with a fair number of tubercle bacilli at their edges.

The *lung* shewed caseous areas of various sizes, but no giant-cells; scattered tubercle bacilli were present at the edges. A little broncho-pneumonia existed around these lesions.

Summary.—The alleged rarity of cavity formation in the lungs of Australian aborigines affected with tuberculosis is upheld by the two cases described. In both cases, solid tubercular lesions were present in various viscera, strongly suggestive of malignant disease in one case, of lymphadenoma in the other. Tubercle bacilli were detected in both cases, being very numerous in parts in one.

DISCUSSION.

Dr. W. A. CHAPPLE : I deem it a privilege to have an opportunity of addressing a distinguished audience of members of the profession to which I have the honour to belong. It is the more a privilege, certainly a greater enjoyment, now that I have descended from the high plane you now occupy and become a Member of Parliament. Still if I have ceased to come into contact with individual patients, perhaps I come more into contact with the nation, and can serve the great science of preventive medicine more in Parliament than before.

I think we are very much indebted to Major CUMMINS for the very comprehensive review of the problem of the distribution of tuberculosis amongst the native races, and the comparisons he has given us. I have had the advantage of being a native medical attendant, and I came into contact with this very problem, and what struck me from clinical

observation was the high degree of susceptibility of the natives. Not only do they contract tuberculosis early, but when they get it it spreads rapidly. Nearly every case of phthisis was one of galloping consumption. General tuberculosis was common, and the rapid spread of the disease led to an early death. Very early in my experience I came to the same conclusion which the author of this paper has arrived at, that the susceptibility was due to tissue vulnerability, owing to the absence of contact with tuberculosis. Not however to its absence in the individual, but to its absence in the race.

If I may mention one word of criticism it is that the author has fixed his attention entirely on the ontogenetic contact with tuberculosis. Phylogenetic contact with tuberculosis will explain the gradual diminution of susceptibility in the white races. Perhaps I should make myself clear, since I am saying this by way of criticism. We must look at this from a biological point of view, and consider the racial rather than the individual history. During generations of contact between the white races and tuberculosis there has been a dying off, in the early years of life, of those most susceptible to the disease. Being highly susceptible, originally they contracted the disease early in life and died off, leaving no progeny. Those less susceptible lived longer and left progeny, and these became the inheritors of a gradually increasing invulnerability. What are the causes of tuberculosis? First, we have that tissue vulnerability, which may be either hereditary or acquired; and secondly, the presence of the organism, which, in the absence of a high degree of tissue resistance, invades, multiplies and spreads. For centuries we have lived in contact with this organism. The fittest to overcome it have survived and left progeny inheriting their fitness. Those least fit have died and left few or none to inherit their defect. Native races have not had this history. I think we have to look then to contact of the race with tuberculosis, in order to explain our gradually decreasing vulnerability to the disease, and conversely we have to look to the absence of any such racial contact in the case of the native races to explain their high degree of susceptibility.

Of course, the presence of the germ, and also the subsidiary causes, such as bad air, bad sanitation, overcrowding, etc., can be guarded against and must be removed, but you cannot remove that phylogenetic susceptibility. It requires centuries of martyrdom to bring this change

about. We therefore owe to the native races a very much higher duty when, in the spread of our otherwise beneficent civilisation, we bring germs to them, in the absence of which they would remain free from disease. This therefore puts upon us, who are empire builders, and who deal with native races in all parts of the world, a very high moral responsibility, and it is our duty to leave no stone unturned to undo the harm we have already done in bringing the germ of tuberculosis into contact with them, and to do what we can to educate and protect them.

I have been frequently struck with the truth of these propositions in relation to the problem of the drinking habit in native races, and I believe that the biological statement I have now made with regard to tuberculosis and the susceptibility of native races to this disease, also holds good with regard to their susceptibility to alcohol. In New Zealand, the Maori race, in the presence of alcohol, take to it with a rapidity that is alarming. When they are placed in contact with alcohol, they shew a high degree of susceptibility. A small dose has a rapid effect, and they quickly become victims to the habit, and suffer from tissue degeneration more rapidly than the whites do under similar circumstances. I need not labour this, but content myself by pointing to the analogy.

We have, therefore, a high moral responsibility with regard to the native races. It is our duty to keep from them the exciting cause of alcoholism, and to realise that we, who have brought the tubercle bacillus, have also brought the drink to them. A grave responsibility rests upon us to protect them from a danger as destructive as the disease we are considering to-night.

Dr. SANDWICH: I should like first of all to say how much the Society is indebted to Major CUMMINS for his masterly paper. It appeals not only to those of us who are present to-night, but will interest our absent Fellows who will have the opportunity of reading it in the Transactions. It is a very long time ago since Egypt began to be associated with the disease we now call tuberculosis. All the early writers speak of it, and CELSUS in the first century recommended patients to change their climate "for instance to quit Italy for Alexandria" in Egypt. I would remind the Fellows that CELSUS like the rest of us began life as a layman, and was so much interested in the study of medicine that he wrote his classical work on the subject.

Nobody can speak with greater authority than Major CUMMINS with regard to tuberculosis in the Egyptian Army, and he has compared in a very interesting way the incidence of the disease between the Sudanese and the Egyptians. It is most interesting to me to find that he has come to the same conclusion to which I arrived with regard to general hospital patients in Cairo—I found, for instance, in 1892 that amongst 300 tubercular hospital patients, all civilians, no less than half of them were Sudanese. This is important because the Sudanese population of Cairo consisted of only 8,000 people. During the same two years the Egyptians furnished 69 cases of tuberculosis, taken from a population of 300,000 in the town itself, besides large numbers from the neighbouring country districts. Then I should like to say a word upon a branch of the subject which Major CUMMINS has not touched upon—the very different way the disease behaves with regard to the two races. For instance, take the duration of the disease in fatal cases. It is quite common in this country, and other parts of Europe, for a doomed patient, infected with tuberculosis, to live about two and a half years after the beginning of the symptoms, and in the United States AUSTIN FLINT found that the patients sometimes lived two and three-quarter years. We can take two and a half years as an average time for white patients to live, even when they are bound to die of the disease. The Egyptians, judging from my experience, lived on an average twelve and a half months, with a minimum of one month and a maximum of five years. The Sudanese average is seven and a half months only. Many of the cases live for only three months, so that the disease is not only more common among black people but is much more serious and more rapidly fatal. I may say it is the only disease of which the Egyptians seem to be afraid. Syphilis, leprosy, and cancer have no horror for them, but the moment they meet with tuberculosis they are apt to lose their equanimity.

Major CUMMINS has spoken of the decrease in the mortality among the monkeys of the Zoological Gardens to which I can also testify. I would only venture to ask him if it is not a fact that about the same time that the monkeys were removed from too close contact with visitors they were given more fresh air. I remember pointing out to Captain FLOWER, Director of the Zoological Gardens in Cairo, that we humans believed in fresh air for tuberculosis, and if he gave the monkeys an open-air life in his gardens, they would not die of tubercle.

I may say in corroboration of this point that patients in the lunatic asylum, which is in the desert outside Cairo, suffered tremendously from tuberculosis. In the old days it was the chief cause of death. When Dr. WARNOCK went there he re-organised the asylum and compelled the lunatics to be outdoors all day, and improved the ventilation by night in the wards, continuing the same diet as before. Before his time the insane, who were not excited, spent all their days sitting on their beds in their sleeping rooms. They never went out. He changed all that, so that in 1910, with an average residence of 1,234 patients, there were 126 deaths, and only 10 of these were due to tuberculosis.

There is one race in Egypt with which Major CUMMINS has not, perhaps, come intimately into contact. I mean the Bedaween, the real inhabitants of the desert. I could never find any trace of tuberculosis in them so long as they lived in the desert, even comparatively near Cairo where civilization exists, and those who visited the Oases in the desert always told me they never discovered any tuberculosis there. But when the Bedaween became, as we should call it, civilised, and married the fellaheen women and came to live in dirty mud huts on the confines of Cairo, then they contracted the disease, whether from the huts or from their brides I am not prepared to say.

An interesting experience occurred to me once. I was asked to find a healthy black boy for a German Princess, to take back to Europe with her as a page. The Crown Princess of Sweden had lately visited Egypt and had returned to Europe with a small boy of the blackest hue. This created envy amongst other Princesses, and one, then visiting Egypt, asked me to find a boy for her. She said the boy must be absolutely healthy, because she had heard that the boy who went to Sweden was suspected of tuberculosis. I examined a good many boys and explained to her that tuberculosis was not the only disease of which Sudanese boys were carriers. It was in the early days of KOCH's tuberculin, which I now know we gave in too large doses, but we thought it was a reliable diagnostic method, and I put several boys through this test and they invariably re-acted. I could not find a healthy black boy whose parents were willing to let him go to Europe. I then suggested to her, how would she like to take one of the Bedaween children, and explained to her that there was much less liability to tuberculosis. This was eventually accomplished with satisfactory results.

I was also interested in Major CUMMINS's suggestion that possibly some day we may vaccinate black people against consumption. That is quite a possibility of the future, I think. I would remind the Fellows of what is already being done in Germany and the United States with regard to the calves to which Major CUMMINS referred. If a calf is born of a tuberculous cow and left with its tuberculous mother the calf will die. If the calf is inoculated with small doses of bovine tuberculin the calf will also die, but if that calf is taken away from its mother and inoculated with small doses of human tuberculin the calf will live and will be able to return to the herd from which it came. It is quite possible that bovine tuberculin may be the solution for which Major CUMMINS is looking. I may mention three cases of tuberculosis of the testicle reported from Egypt by Dr. HADGES. Two Jew babies were circumcised and both died from general tuberculosis. They were both operated on by a tubercular Rabbi, and, as everyone knows, in the operation of circumcision, as carried out by the Jews, the hæmorrhage is arrested by applying the mouth of the Rabbi to the injured part. A third case was a Mussulman boy, circumcised at the age of seven years by a barber who had tuberculosis of the left index finger. The cases are sufficiently rare, perhaps, to justify their mention.

I should like, in conclusion, to say that during the 23 years which I spent in Egypt, I met of course with a large number of people who came there for their health, many tubercular, but I only met with one English person, I can almost say one European, of all my acquaintance who contracted tuberculosis of the lungs in Egypt. That is a very extraordinary result, I think, unless I was extra fortunate in my acquaintances.

To-night we have spoken chiefly of tuberculosis of the lungs; however, I think we can say that both medical tuberculosis and surgical tuberculosis supply something like one fourth of the patients who died in the Kasr el Ainy hospital in Cairo.

Dr. DANIELS: I was very much interested in this question of tuberculosis, and for two reasons. One is that it is a disease which occurs in many parts of the world and carries off more patients than any other individual disease, and the other is that I have had the misfortune to be in two countries that were nearly free from tuberculosis as regards the natives, and subsequently it has been introduced, and on being introduced

has spread rapidly. First of all, in Fiji, where I was in 1889. Just before I left I had come across a single case of tuberculosis amongst the natives, neither the medical officers nor the natives having ever seen any before except amongst imported coolies—Calcutta coolies and Melanesians. Since then tuberculosis has become quite common, not only amongst the natives but amongst Europeans. Two medical officers who went out since then acquired the disease and died of it.

Another place—Central Africa. In 1899 tuberculosis was unknown amongst the natives. Since the war, many of the natives, attracted by the higher wages in the Transvaal and Southern Rhodesia, have gone to the mines, acquired tuberculosis and come back with it. The disease is very common there now, where before it was quite unknown, and there again it has spread to the Europeans, so that they in their turn get infected. These cases are within my own knowledge.

In British Guiana, about 1847, we have records of some 1,400 post-mortems, and 29 cases of tuberculosis were noted amongst the 1,400, a special note being made that everyone of these cases was that of a person who was not a native of the colony, but was an importation from Madeira or elsewhere. There were no native cases.

Of the post-mortems I made in 1902-1905, in the same country, over 25 per cent. of the adult cases were cases of tuberculosis, and tuberculosis was the cause of death, that is to say it was in an advanced or acute stage, and the proportion amongst the natives of the colony was rather higher than among the other races. All this points to the great importance of the subject and the enormous harm that has been done by the movement of natives. How to prevent contact, I do not know. You have a primitive race like those in Fiji, and when the sugar industry started, in order to get the people to work, it was necessary to employ tuberculous races. You have mines, and must have native labour, or Chinese labour, which is already more or less immune to about the same extent as the Europeans. Chinese labour has gone, and you therefore have to import black labour, who will get tuberculosis and take it back to the districts from which they came. Whether it is sugar, or rubber, or coal, or gold mines, or any other mines, as long as you have these conditions you will disseminate tuberculosis to the previously healthy peoples.

I cannot say I feel very sanguine about the possibility of immunising the individual or of individual recovery from tuberculosis. I quite agree

that as a race we may claim a certain degree of immunity, but you must take into account the extraordinary rapidity of the spread of the disease amongst the native races. The majority of the negroes, and it is the same with other primitive races, die after a short febrile illness, before physical signs have yet developed; cavity formations do not occur, the man does not live long enough for that. I have seen fatal cases in much less than three months. It is not a case of getting it as children; you will find a good many cases who die in the first year of life, so that there is no chance there of any immunising of the adult, unless it can be done artificially with cultures of tubercle bacillus whose virulence has been reduced—but that is a matter of considerable expense, and at present only a theoretical possibility—and isolation.

One other point against the possibility of immunising the individual. Amongst the post-mortems I have done are Europeans, Chinese, and Indians, and amongst all these races you will find a very fair proportion of cases who die from dysentery, malaria, or accident. You will find any amount of evidence of old healed tuberculosis, but amongst the negroes never. The negro either has tuberculosis and dies of it, or at the time he dies he has it at the acute stage and not the healed stage. These, to my mind, are strong arguments against the immunisation of the individual.

Dr. LAW: I would like to mention just one point which Dr. DANIELS has not mentioned with regard to the West Indies, the less frequent appearance of bone tuberculosis in the black races than in the white races. You do not find bone tuberculosis as common in the African races of the West Indies as in the people of this country. This is one point Dr. DANIELS did not mention. I think one thing we can do is to teach them the value of ventilation. It has been said that tuberculosis, which was almost unknown in the times of slavery, increased as the black races became free and were able to house themselves. In the old days of slavery, they were more or less taken care of; there was any amount of natural ventilation in their houses, but when they became free and lived in wooden houses, they stopped up every possible chink where the air could come in at night, and tuberculosis increased. Of course there is no doubt there is a peculiar susceptibility of the race, but if they were taught to ventilate their houses and live in the fresh air,

their susceptibility to tuberculosis would considerably lessen, and by degrees they would come to the condition we are now in of being much less susceptible to the disease.

There is an analogy in the same way with syphilis. The black races were not subject to syphilis during slavery, great care was taken of them, but when they became free they became infected with that disease also, and the manifestations were much more severe than those seen in this country. It is just a parallel case, and appears to indicate that a race by long exposure to any particular disease acquires a certain degree of resistance to it.

Mr. AUSTEN: A point to which I would like to refer is the question of the occurrence of tuberculosis in the anthropoid apes in the wild state. Major CUMMINS says monkeys in the wild state appear not to suffer at all. He also points out the marked susceptibility of apes to tuberculosis in captivity, but he does not say anything about their susceptibility in the wild state. Some years ago I was informed by the late Col. C. T. BINGHAM that in the forests of Burma Gibbons suffer to a very large extent from tuberculosis. Col. BINGHAM said one can hear them coughing in the trees; they cough, cough, cough away, and appear to be suffering from tuberculosis of the lungs. Well, BINGHAM was not a medical man, and did not apply any tuberculin test or anything of the sort, but I do not think it altogether likely that he was mistaken. He was a Conservator of Forests in Burma for upwards of twenty years, and was a competent and trained observer, being for some years Editor of the volumes published by the Indian Government on the "Fauna of India," and he certainly knew what he was talking about. Two species of Gibbon are found in Burma—the Hulok Gibbon, of which the range extends from Assam to the Irrawaddy river, and the White-handed Gibbon in Lower Burma. Which species it was I do not know, but it certainly was one of these two. Col. BINGHAM is unfortunately now dead, so that no further information is obtainable from the original source; but I thought it might possibly be worth while to mention his statement, so that the attention of some officer of the Indian Medical Service or other Fellow of the Society stationed in Burma might be directed to the matter. If wild animals such as Gibbons, living in a state of nature and leading an almost entirely arboreal existence, really do suffer from phthisis, the point would seem to

be worth considering, if only as affording a remarkable contrast to the "virginity of the soil" in this respect observed by Major CUMMINS among primitive races in Africa.

Sir HAVELOCK CHARLES: It appears to me that the spirit of the debate suggests:—First, the very powerful influence of the "virgin soil"—this is unanimous. Secondly, that most Europeans have been subjects of tubercle at some time or other. Thirdly, that the European has elaborated a protective principle which confers partial immunity. Fourthly, there are certain races which possess no immunity against tubercle.

Authorities can be quoted for the assertions that this is one of the oldest diseases, that no single climate grants immunity, that it is known to occur in all regions, that of all infectious diseases it causes the highest mortality, that no race, no geographic site is exempt from it. The vital points in the development of the disease are overcrowding and unhealthy occupation. Observation shows that there are some races that have a certain immunity to the toxæmia of tuberculosis and such patients withstand the disease for a long time. This points to an inherent resistance. Such patients in my experience were Jews.

Reference has been made to the incidence of tuberculosis in the Indian Army. May I point out that the admission and death-rate have fallen enormously. In 1901, the admission rate for tuberculosis for the Indian Army was 4·2 per 1,000; in 1910 it was 2·4. The death-rate for the same period had fallen from '8 to '1 per 1,000. The peculiar vulnerability of the Gurkhas has been referred to. Here again I wish to point out that, with improved conditions in housing and training, the admission rate for tuberculosis amongst them has fallen from 13·1 per 1,000 in 1901 to 3·6 per 1,000 in 1910, whilst the death-rate per 1,000 for the same period has diminished from 3·9 to '5! The Gurkhas also no longer head the list with the largest number of cases. In 1910, the Guides had 17 admissions. The 76 Punjabis in North China had 15 admissions and the 212 Gurkhas (Derha Dhun) had 9.

The Guides are one of the finest regiments in the Indian Army. It is reported that nearly all the cases I mention became infected at Fort Abazai, where the ventilation is bad and the lighting very insufficient. There are no Gurkhas in the Guides. Life and work in barracks,

infected from old time, with limited space, and bad ventilation must tell—no matter what class of men may compose the regiment. I have pointed out that modern sanitation, and attention to it, is already altering the previous high tubercle admission rate and mortality of the Gurkha.

The protection of so-called susceptible races depends on sanitation. The Indian Army furnishes this lesson, and we have had object lessons on the subject amongst the Garrison in London.

The London Zoo has been mentioned. There again improvement in sanitation has been the cause of a great decline in the incidence of tuberculosis. In 1908, there were 5,608 mammals, and there were 58 deaths due to tubercle. In 1911, with 6,677 mammals in the garden the deaths due to tubercle had fallen to 18. Facts like these bring the lesson home.

Much has been spoken of the negro in the U.S.A. He is said to have no chance now when he contracts tubercle. Why? Let us review his career. Before the war he was a valuable chattel, the care of his master, with good food, living in the open air, under the discipline of regular healthy labour and suitably clad. After the war, we have this race, immature mentally and morally, given its freedom. What followed? Released from the country, they herded in large cities, leading lives of comparative idleness, victims to intemperance and excessive venery, with bad food, bad air and weakened by the ravages of syphilis. Does it require any theory of virgin soil to explain the resistless course of the disease amongst them? There should be no difference of opinion regarding the conditions of life that sap vitality, and so directly predispose to the hundred-fold crop of tuberculosis. If you have overcrowding, whether in private dwellings, workshops or barracks, and the pursuit of insanitary occupations, and these combined with idleness, intemperance, bad or deficient food, and evil living, you cannot by any means in the present age protect any race or nation from the ravages of tuberculosis once the bacillus gains a footing.

Major CUMMINS: Mr. President, I should like to thank you and the Fellows of the Society for the very interesting discussion in which so many have taken part. I think a great deal of light has been thrown on the subject. There are so many points which have been raised that

I cannot hope even to attempt to reply to them all. To begin with : Dr. CHAPPLE, in his very interesting remarks about New Zealand, touched upon heredity. There of course one gets into a subject which is very deep and difficult. I think myself that one need not go to heredity to explain the facts, but very likely further research will prove such a theory to be necessary, and I can see there may be a great many reasons in favour of it. For instance, in the case of the American negroes it may be claimed that on account of their contact with civilised people for so long a time, they ought to have acquired immunity if it is to be acquired. One can explain their not having done so by saying they have an hereditary tendency to tuberculosis, but I think one can also explain it without committing oneself to such a difficult hypothesis. The negro slaves were well looked after and well sanitated, and when they became their own masters this was no longer the case. Under conditions of over-crowded town life they were more in contact with whites, and liable to large doses of tubercle bacilli, against which they were not protected. It is a most interesting speculation whether protection can be transmitted or not. It would be presumptuous for me to attempt to deal with such a question as the transmission of acquired characters, but many people in a position to form an opinion believe such a transmission to be possible.

As to Dr. SANDWICH's interesting remarks about Cairo, his long experience in Egypt enables him to speak with authority, and also Mr. MILTON. All they say confirms, with their wide experience, what my limited experience has led me to believe. In the case of Egyptians, although they get tuberculosis, still they often recover ; but amongst the Soudanese the disease nearly always runs a rapid and fatal course.

I will go straight on to the remarks of Sir HAVELOCK CHARLES. I think we agree in all the essentials. I am quite sure that the great hope at present lies in improved sanitation, and how does sanitation act ? If you can lessen the density of humanity on a given area, for instance in a room, and at the same time increase the amount of, and the interchange of, fresh air, you at once diminish the liability of the inhabitants to a lethal dose of tubercle bacilli. You give them more healthy surroundings, and at the same time you give them an immunising instead of a fatal dose.

I am very glad to hear of the statistics referring to the Gurkhas.

It is more than interesting to hear that under conditions where a large and fatal dose is impossible, instead of having a high death-rate they have only a small incidence.

The question of protection by vaccines is one that will come to the front more in the future. It has recently been discussed, I believe, at the Congress in Rome. Several people, I understand, have brought forward reports which appear to show that protection against tuberculosis by means of vaccines is going to become an important factor in the combat against the disease. I think myself it is bound to do so. Having had a comparatively large experience of work in typhoid prophylaxis, perhaps I am rather prejudiced in favour of vaccines.

As to the Gibbons, to which Mr. AUSTEN referred, his remarks are very interesting, and it is a question upon which I hope, sooner or later, information will be forthcoming. If the Gibbons are very liable to tuberculosis in their wild state one would expect some of them to bring it with them to the Zoo, but the fact is that no anthropoid has died of tuberculosis for some time past. It should also be borne in mind that the sanitation at the Zoo has been considerably improved.

In the old days animals were often brought in in bad health, in hopes that proper care would enable them to improve. Nowadays the animals are inspected, and any who present any symptoms of tuberculosis are rejected. I think perhaps this diminished contact with tubercle bacilli explains why these highly susceptible animals now contract the disease in such comparatively small numbers.

PELLAGRA: SOME NOTES ON ITS EPIDEMIOLOGY IN AMERICA.

BY R. M. GRIMM, A.M., M.D.,

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The purpose of this article is to present a few facts which were collected by me during the summer of 1911, while making studies in the epidemiology of Pellagra in the United States. These studies were made under the orders of the Surgeon-General of the United States Public Health and Marine-Hospital Service, and represent a part of the work which this Service is doing on the subject of pellagra. My official reports have already been submitted and some of them have been published,* but this article brings together for the first time the results of my epidemiologic work done during the summer of 1911. The purpose of the work in which I was engaged was primarily to collect facts of an epidemiologic nature, and incidentally to attempt to discover some promising line along which more intensive studies could be made. Since these studies were largely preliminary no conclusions have been drawn, but the following facts and observations are presented here in the hope that they may be of some interest now, and of possible value in the future when supplemented by further work.

This article is based on facts pertaining to 500 cases of pellagra. The number includes 120 cases collected in the State of Kentucky (Series WKB) and 380 cases in the State of South Carolina (Series SCY). Pellagrous districts in three counties in each state were visited, and as much information collected in each district as circumstances would allow. Whenever possible the pellagrins were visited at their homes; and when this was not possible the local physicians were depended upon for the information. These 500 cases do not represent the total number of

* *Public Health Reports*, February 23rd, 1912. *The Lancet-Clinic*, Cincinnati, U.S.A., March 2nd, 1912.

pellagrins in the six counties, but do represent the large majority of the cases of pellagra which have occurred in the districts which were visited. The total number for the six counties would undoubtedly greatly exceed 500.

The two sections of country in which these cases occurred differ somewhat in topography. The cases of Series WKB occurred in the mountainous coal-mining section of south-eastern Kentucky, and the cases of Series SCY occurred in the rolling and hilly cotton-growing section of north-eastern South Carolina. The former section is on the western slope of the Allegheny Mountains and has an average elevation of about 1,000 feet above sea level, while the latter is located to the eastward of these mountains and has an average elevation of about 600 feet. Both sections have good natural drainage and are in many respects ideal places to live. The climate is temperate and never extreme. There are many streams, varying in size, and usually swiftly flowing, as they wind their way among the many hills. Springs are numerous in both sections, some flowing boldly throughout the year, others varying in flow and perhaps becoming dry during the summer. The inhabitants of the Kentucky districts which were visited depend largely upon the coal-mining industry, and it was in the coal-mining camps that many of the cases in Series WKB were found. The South Carolina districts are largely agricultural ones, and the growing and weaving of cotton are important industries. There are many cotton mills in this section, and many of the cases in Series SCY were found living in the little villages located around the cotton mills. The inhabitants of these sections are whites and negroes. The negroes in the Kentucky districts are in the minority, but in the South Carolina districts they were found more numerous, and in some communities almost equalled the whites in numbers.

For collecting and filing the data 5in. by 8in. cards were used. These proved fairly convenient and satisfactory. The face and reverse sides of the card are shown here and need no explanation. See *Fig. 1* and *Fig. 2* (pp. 292, 293). A supplementary blank card was used for additional notes and remarks. Each case was given a card. The cards were then numbered and arranged in consecutive order in the series.

The collection of data of this kind is a slow and laborious procedure, and necessitates constant tact on the part of the epidemiologist in order to obtain the desired information. In many instances my notes are far

from complete, but after all considerable information on the epidemiology of pellagra has been obtained.

CONSIDERATION OF THE TOTAL NUMBER OF CASES.

Under this heading are given a few facts which apply both to the cases personally seen and to the cases reported to me. The notes on the reported cases are necessarily briefer than those on the cases seen, and the facts taken from them are given here along with the corresponding facts pertaining to the cases seen. The cases and deaths in the two series are given below:—

	Series WKB.		Series SCY.		Totals.
Cases seen ...	38	...	83	...	121
Cases reported ...	64	...	208	...	272
Deaths reported...	18	...	89	...	107
Totals ...	120		380		500

With reference to race and sex these cases have been grouped as follows:—

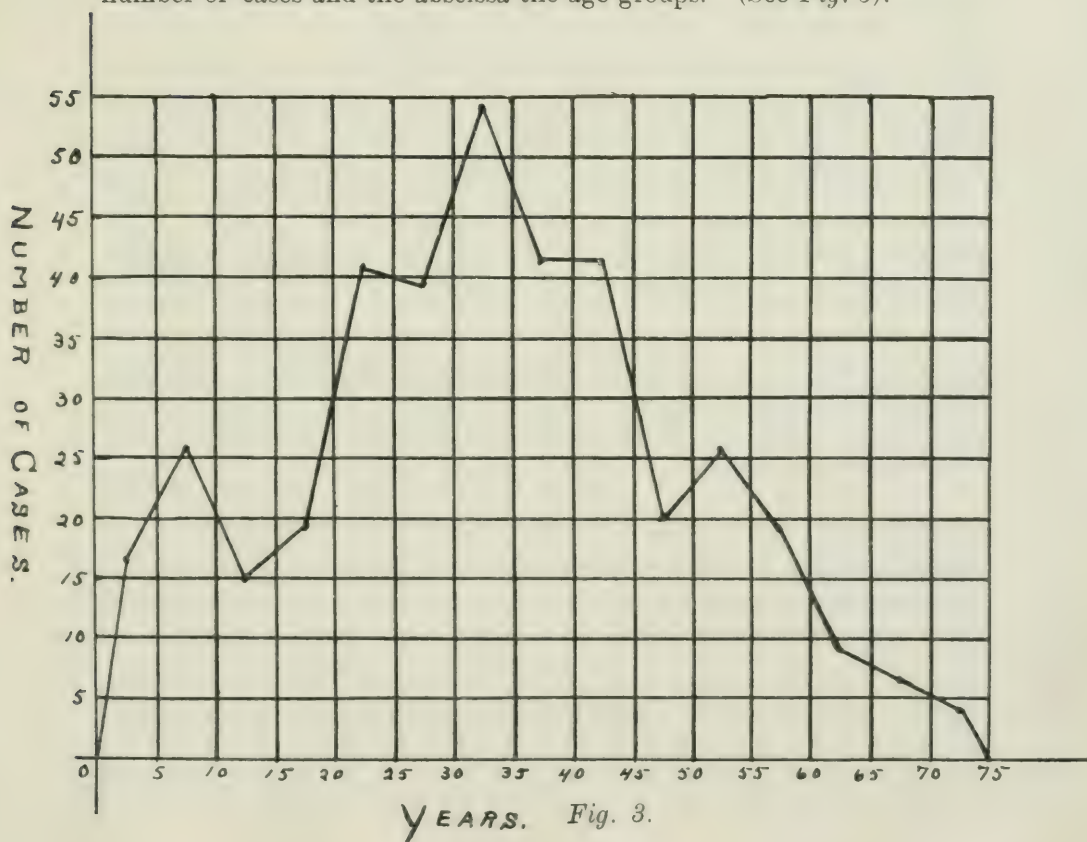
	Cases seen.		Cases reported.		Deaths reported.		Totals.		Percentage of whole.
White females ...	87	...	180	...	70	...	336	...	67·2
White males ...	30	...	67	...	21	...	119	...	23·8
Coloured females...	2	...	13	...	11	...	26	...	5·2
Coloured males ...	2	...	4	...	5	...	11	...	2·2
Sex not reported...	0	...	8	...	0	...	8	...	1·6
Totals ...	121		272		107		500		100·0

With reference to age these cases have been grouped as follows:—

Age group.	Number seen.		Number reported.		Total.
0 to 4 years ...	5	...	12	...	17
5 to 9 „ ...	11	...	15	...	26
10 to 14 „ ...	3	...	12	...	15
15 to 19 „ ...	7	...	12	...	19
20 to 24 „ ...	19	...	22	...	41
25 to 29 „ ...	12	...	27	...	39
30 to 34 „ ...	14	...	40	...	54
35 to 39 „ ...	14	...	28	...	42

Age group.	Number seen.		Number reported.		Total.
40 to 44 years ...	8	...	34	...	42
45 to 49 „ ...	7	...	13	...	20
50 to 54 „ ...	9	...	17	...	26
55 to 59 „ ...	6	...	13	...	19
60 to 64 „ ...	2	...	7	...	9
65 to 69 „ ...	3	...	4	...	7
70 to 74 „ ...	1	...	3	...	4
Above 74 „ ...	0	...	0	...	0
Not reported ...	0	...	120	...	120
Totals ...	121		379		500

The 380 cases in the column of totals in which the age was determined may be expressed graphically by a curve where the ordinate measures the number of cases and the abscissa the age groups. (See *Fig. 3*).



Marital Condition.—This was determined in all of the cases visited, but not in all of the reported cases. The numbers as learned are as follows:—

Married	...	...	...	265
Single	...	...	...	103
Widowed	...	...	...	13
Not reported...	...	...	...	119
Total				500

Of the 265 married pellagrins, 238 were married white females. Of the 103 single ones, 57 were children under 15 years of age.

Mortality and Insanity Rates.—As has been said, there are 107 deaths included in the 500 cases. These are practically all of the deaths from pellagra diagnosed as such that had occurred in the districts from which these cases were collected. This gives a mortality rate for the 500 cases up to the present time of 21·4 per cent. In regard to the incidence of insanity in the whole number it was found that 24 had become insane after the onset of the pellagrous symptoms. Some of these had been confined, at least for a time, in insane asylums. This gives an insanity rate for the 500 cases of 4·8 per cent. Both of these rates are undoubtedly low for the disease, since the great majority of these cases have not yet been followed to the bitter end.

Date of Onset.—On account of the fact that pellagra has been for a long time unrecognised in the United States, it could not be determined with any degree of accuracy the time at which pellagra appeared in the two sections which were visited. Judging, however, from the histories of some of the cases in this series, the survivors as it were, it would seem that the disease had been present here for a number of years. It appears also that, since the recognition of the disease here, each successive year has furnished a larger number of cases than the preceding year, and that more cases are now being recognised in these two sections than ever before. The year of onset of the disease was determined in 308 of the 500 cases, but these, it must also be remembered again, are only the survivors. The numbers having their onset in the several years are as follows:—

Year of Onset.				Number.
1911	...	...	...	156
1910	...	...	...	74
1909	...	...	...	30
1908	...	...	...	13
1907	...	...	...	9
1906	...	...	...	7
1905	...	...	...	5
1904	...	...	...	6
1903	...	...	...	3
1902	...	...	...	1
1897 (?)	...	...	...	1
1893 (?)	...	...	...	2
" Many years ago "	...	...	...	1
Total				308

CONSIDERATION OF CASES SEEN.

Under this heading are given those facts which were collected at the visits and which have not been given under the above heading. I regret that I was not able to give each case a more careful consideration, but the difficulties encountered in such work are great. The clinical side of the cases could not be gone into with any detail, and after getting enough of the history necessary for a positive diagnosis, the time was spent in getting the data pertaining to the epidemiology. The cases considered under this heading are 121 cases which were seen. Practically all of these were seen at their homes, but only 56 of this number were found living at the houses in which they developed the first symptoms of the disease.

Environment and Conditions.—As has been said, the cases of Series WKB occurred in the mountainous section of south-eastern Kentucky and those of Series SCY occurred in the upland section of north-eastern South Carolina. Many of the first series were found living in the coal-mining camps of the former section, and many of the second series were found living in the cotton-mill villages of the latter. The general living conditions of the people in these two classes are in many respects identical. Both classes are largely dependent upon the industrial companies

employing them. They live in the companies' houses and obtain much of their food from the companies' stores. This statement does not apply to all, but to the great majority of these people. Many of them are ignorant and live a hand-to-mouth existence. Their incomes are generally not sufficient to furnish the necessary quality and quantity of food. It was learned that in many of these homes, even when it is obtained in sufficient quantity, the food receives poor culinary preparation. These classes seem to be dissatisfied and restless, and are nomadically moving either from village to village or from house to house in the same village. This fact was found to complicate matters greatly.

The class of cotton-mill people among which so many of these cases occurred seemed to be generally poor, overworked, and underfed. They were found living in little cottages located around the cotton mills in which they were employed. These villages are usually looked after by the mill operators, but the authority of the operators does not extend to the interior of these homes, where in many instances were found the signs of poverty and extreme neglect. With the exception of a very few of the homes which were visited among this class, it can be said that the general hygienic condition of the houses was uniformly bad. Specimens of the ordinary bed-bug were collected from some of the homes by a tactful family physician, and in very few of them, indeed, were conditions such as to lead one to suspect the absence of this parasite. The great majority of the pellagrins in this class were found living in unscreened houses, although mosquitoes were said to be plentiful in several of the communities.

The hygienic conditions of the homes visited among the coal miners were about the same as, if not worse than, those found among the cotton-mill class. The coal-mining camps visited were comparatively new ones, and conditions were found which usually accompany the starting of new camps in comparatively isolated spots. It is practically impossible to keep these camps in good sanitary condition. Dirt, trash, and refuse of various kinds were found scattered around the premises. One of the camps was having a small epidemic of typhoid fever at the time of my visit. The conditions of living in these camps are continually improving, however, as the mine operators are keenly interested in the health of their employees and are constantly on the lookout to improve them.

Of the pellagrins not in the cotton-mill or coal-mining classes there were found three female pellagrins who seemed to be in good economic circumstances, to be living under good sanitary conditions, and who appeared to be neat and cleanly housekeepers.

As to the economic condition of the 121 cases, three were considered good, two moderate, and the remainder poor. The sanitary conditions of the homes were found good in three, fair in two, and bad in the remainder. Some of these were extremely bad.

Location of Homes.—With reference to this point the following were found:—

In a cotton-mill village...	...	...	70
In a coal-mining camp ...	...	...	24
In a town ...	...	...	14
In the country ...	...	...	13
Total ...	...	...	121

In regard to the proximity to streams, all of the homes of pellagrins personally visited were found located within half a mile of some running water. The great majority of them were much nearer than this; and some were literally on the banks of streams. All of the coal-mining camps visited were located along some creek or run, and this was the common finding with reference to the cotton-mill villages. Some of the cases reported to me were said to be living at greater distances from running streams, but the exact distances were not learned.

Occupation.—The occupations of the 121 cases seen are as follows:—

Cotton-mill people—

Cotton-mill workers	...	...	17
Housework	...	...	38
Children ...	...	...	12

Coal-mining people—

Coal miners	...	...	4
Housework	...	...	14
Children ...	...	...	6
Book-keeper	...	...	1

Agricultural people—

Farmers ...	...	...	2
Housework	...	...	6

Railroad people—

Railroad man	...	...	...	1
Children ...	...	...	...	1

Labouring (miscellaneous)—

Labourers	...	...	...	2
Housework	...	...	...	17

Total	...	...	...	121
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Food.—The subject of diet in these cases, although a very promising field for careful study, could not be gone into thoroughly under the circumstances. The question of food was found to be varied and complex. The sources of food supply, too, were varied in many cases. The pellagrins of the coal-mining and cotton-mill classes had been obtaining much of their food from the stores of the companies employing them or some member of their family. The majority of this had been imported and was in the shape of canned goods, packed meats and the like. It seemed that very little of the food used in these families had been home grown. Fresh vegetables and fresh meats had been used only to a limited extent. The stores supplying food to these people are run by the companies both for the convenience of their employees and for their own profit. A list of the articles most sold at one of these stores was given me by a storekeeper. The list is given here because it is rather typical: bacon, lard, flour, cabbage, potatoes, onions, rice, pie peaches, table peaches, dried peaches, dried apples, canned corn, canned tomatoes, canned peas, canned salmon, kraut, hominy, blackberries, corned beef, canned roast beef, potted ham, sausage, canned pumpkins, canned cherries, canned sweet potatoes, postum, coffee, tea, sugar, baking powder and soda. This list is typical of the stock carried by these stores, although many of the families patronise stores which carry a better and more varied line of articles. The subject of diet certainly seems to be a most promising one, and it is hoped that further and more thorough studies can be made along this line.

Each one of the pellagrins who were seen gave a history of the more or less regular use of corn in one form or another as an article of diet. Many of them stated to me that they "had lived on corn bread." Corn in various forms constitutes one of the main articles of diet of the people

throughout both of the sections in which these cases occurred, and the people seem to feel that they cannot get along without this cereal. For these people to have vegetables on the table without corn bread is contrary to a firmly established custom, and not at all in accordance with their taste. On account of the passing of the old-time grist mill, to which the farmer took his corn at frequent intervals and had it ground, much of the corn meal now used in these sections is not from the home-grown product, but is that which has been ground at some of the large mills of the country, and which, as the people said, had been "shipped in." Considerable time often elapses between the grinding and the consumption of it. Several lots of this meal which had been received at some of the stores were found to show a high degree of acidity, and had been condemned. In several instances the pellagrins told me that the corn meal which they had been using was not of the best quality.

Water Supply.—The sources of the drinking water used in the families of the pellagrins visited were found to be as follows:—

Wells:—Driven	...	...	...	33
Dug	...	...	...	25
City supply (surface water)	...	...	...	7
Well and city supply	...	...	...	6
Creek	...	...	...	2
Well and creek	...	...	...	4
Well, creek and spring	...	...	...	2
Creek and spring	...	...	...	2
Well and spring	...	...	...	9
Spring only	...	...	...	15
Hydrant (fed by mountain springs)	...	...	...	3
Varied	...	...	...	13
Total				121

These sources were inspected whenever possible, but in no instance was a chemical or a bacteriological examination of the water made. In many cases the water was by no means above suspicion. The springs which were inspected were certainly questionable as proper sources of water for drinking. Most of them were located favourably to receive surface drainage, and were found to contain decaying leaves, sticks and

trash of various kinds. Lizards, bugs and other water forms were noted in some of them. A large number of the pellagrins, however, were using water from sources which seemed apparently all right.

Heredity.—None of the data pertaining to this series of cases seems to have a positive bearing on this point. The pellagrous mothers and pellagrous children are grouped below with reference to the condition of their children and mothers respectively. Only the pellagrous children under ten years of age are considered here, and of the pellagrous mothers only those now having children born at or after their first attack of pellagra. None of the pellagrous fathers had children with pellagra.

(A) Pellagrous mothers:—

(a) With healthy children	...	...	17
(b) With poorly nourished children	...	...	2
(c) With pellagrous children	...	...	3

(B) Pellagrous children:

(a) With healthy mothers	...	...	13
(b) With pellagrous mothers	...	...	3*

Domestic Animals.—No relation could be traced between any of the cases of pellagra considered here and any of the domestic animals or the diseases occurring among them. The one family that had four cases kept four goats which were apparently healthy.

Relation of Cases to other Cases.—Questions bearing on this point were asked in each instance and the data collected proved to be varied. These relations cannot be given in detail, but a classification of them is given here, which is fairly inclusive.

(A) Case relations in the family (in same house):—

(a) Families having 1 case	...	...	97
(b) Families having 2 cases	...	...	9
(c) Families having 3 cases	...	...	3
(d) Families having 4 cases	...	...	1

* Ages 4, 7 and 7 years at onset of first attack. Mothers healthy at the time of the birth of these children. Same as "c" in group "A."

(B) Miscellaneous relations :—

(a) No known relation to another case of pellagra	...	...	...	30
(b) Previous to onset having had temporary association with a pellagrin	...	...	...	10
(c) Onset while living in the immediate vicinity with a pellagrin	...	...	...	56
(d) Onset while living in a house with a pellagrin	...	...	...	18*
(e) Onset while living in a house previously occupied by a pellagrin of another family	...	...	...	7

CONSIDERATION OF TWO PELLAGROUS COMMUNITIES.

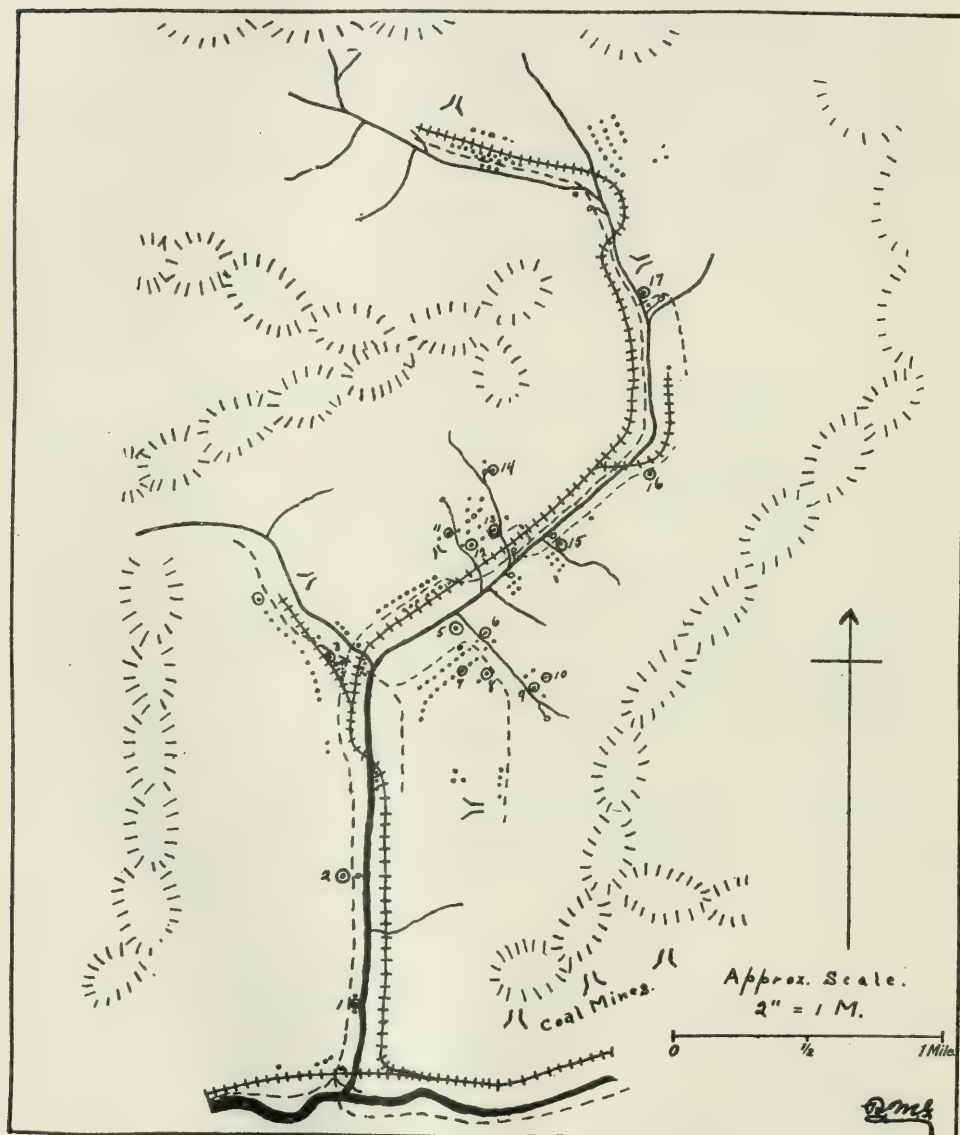
Special attention was paid to a small coal-mining district in Kentucky and to a small cotton-mill village in South Carolina, on account of the comparatively large number of pellagrins who had developed the disease while living there. Rough maps of these communities are given here, which shew the prevalence of the disease in these places and some other points of interest. The maps are in a way self-explanatory. The dots represent the locations of homes and the dots inclosed by circles the locations of homes of pellagrins. The pellagrous homes have been numbered for the sake of reference, the numbers referring to notes accompanying each map. The maps have been designated "District SC" and "Village EM." The cases of pellagra which occurred in District SC belong to my series WKB, and the cases which occurred in Village EM to my series SCY.

District SC is one of the coal-mining districts of Kentucky. The population of the area shown in the map is probably two thousand or more, and practically all of these people belong to the coal-mining class. The coal mines here are the sole source of income of these people, none of them paying any attention to agriculture. Very few of them, indeed, even have gardens to supply fresh vegetables for their tables. There are a few negroes here, but the great majority of the inhabitants of this district are whites. Pellagra was first recognised here in the early

* Included in group "A."

summer of 1911, but in the opinion of the local doctors it had been present here for some time before this. Exactly how long I was unable to determine.

"District SC."



- | | | |
|--------------------|--------------------------|--------------|
| ~~~~~ = Streams. | o = Wells. | o = Springs. |
| --- = Wagon Roads. | • = Residences. | |
| ++++ = Railroad | ⊙ = Homes of Pellagrins. | |

NOTES ON THE HOMES OF PELLAGRINS IN "DISTRICT SC."

1. Case 24 now living here. Developed pellagra here in May, 1911.
2. Case 14 developed pellagra while living here in July, 1909. Later moved to house 3.

Case 6 and Case 7 (mother and son) developed pellagra in the summer of 1911 while living here. Two other children are probable cases.

3. Case 14 now living here. Seen while in third attack.
4. Case 16 now living here. Developed pellagra in July, 1911, while living in house 12.

(No case of pellagra has been known to develop in the little group of houses in the angle of the stream around the railroad switch).

5. Case 13 now living here. Onset in July, 1911, while living in this vicinity.

6. Case 126, a probable case, now living here.
7. Case 11 now living here. Onset in July, 1911, while living here.
8. Case 10 now living here. Onset in summer 1909, while living further up the creek.

9. Case 122 lived here. Later Case 126 lived here just previous to onset of probable symptoms in summer 1911.

10. Case 12 now living here. Developed pellagra here in July, 1911.
11. Case 18 now living here. Developed pellagra here in June, 1911.
12. Case 16 developed pellagra here in July, 1911. Now living in house 4.

13. Case 17 now living here. Developed pellagra elsewhere.
14. Case 19 and Case 36 (sisters-in-law) developed pellagra here in May, 1911. Case 19 now living here.

15. Case 125 developed pellagra while living here. Moved away and died of pellagra in June, 1911.

Case 124 later moved here and developed pellagra here in spring of 1911. Now living here.

16. Case 35 now living here.
17. Case 127 developed pellagra here. Family moved away and the patient died of pellagra in July, 1911.

Case 20 of another family developed pellagra here in the spring of 1911. A probable case in the same family is now living here with Case 20.

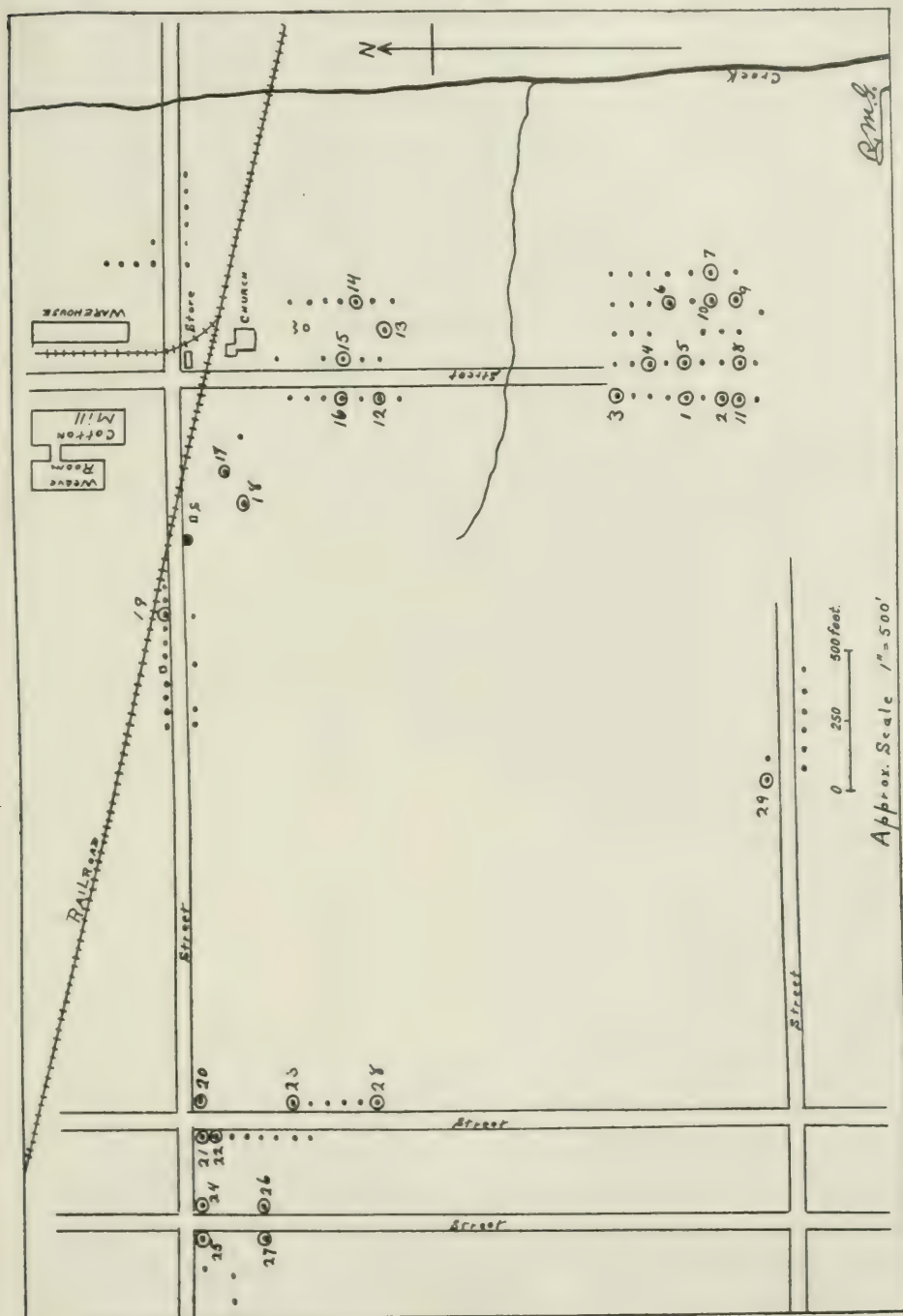
It was learned that the food used in these families had been obtained chiefly from near-by commissaries. A list of the articles most used has been given above. Corn products had been used extensively by all of these pellagrins as well as by the other members of their families. Some of these people told me that the corn meal that they had been using was not always of the best quality. The mining companies have sunk deep wells at various places, but the great majority of the people use surface water from the streams or get their drinking water from springs. The springs which were inspected here were not above suspicion as sources of drinking water. The conditions of the interior of many of these houses were such as to make one suspect that they offered a good field for the collection and study of some of the ordinary ectoparasites of man. The Entomologist of the State University at Lexington, Kentucky, visited this district after me and made entomological collections. I have learned that he found among his specimens some pupæ of the fly *Simulium*.

The map designated "Village EM" shows roughly the plan of one of the small cotton-mill villages of South Carolina. The location of the homes of the pellagrins is designated in the same manner as in the preceding map. The eastern portion of the map represents the cotton-mill village proper, while the western portion represents a part of the suburbs of a neighbouring town. The population of the area shown in the map is approximately 500. The two groups of houses south of the mill are situated on slight elevations separated by a small stream which flows into a larger one not far distant. I was told that no case of pellagra had been known to develop in any of the families occupying the little group of houses in the north-eastern portion of the map. The homes of pellagrins have been numbered as before and refer to the notes given below:—

NOTES ON THE HOMES OF PELLAGRINS IN "VILLAGE EM."

1. Case 250 developed pellagra here in 1904. Died.
- Case 257, of another family, developed pellagra here in 1911.
2. Case 249 developed pellagra here in 1904. Died.
3. Case 227 and Case 228 (brothers) developed pellagra here in 1909.

"Village EM."



Case 254 developed pellagra here in 1911.

4. Case 205 developed pellagra here in 1909.
5. Case 253 developed pellagra here in 1909.
6. Case 252 developed pellagra here in 1910.
7. Case 251 developed pellagra here in 1910.
8. Case 251 lived here after the onset of the disease.
9. Case 214 developed pellagra here in 1911.
10. Case 214 lived here after the onset of the disease.
11. Case 225 developed pellagra here in 1911.
12. Case 204 developed pellagra here in 1910.
13. Case 255 developed pellagra here in 1910.
14. Case 231 developed pellagra here in 1910.
15. Case 256 developed pellagra here in 1910.
16. Case 203 developed pellagra here in 1911.
17. Case 207 developed pellagra here in 1910.
18. Case 223 developed pellagra here in 1911.
19. Case 206 developed pellagra here in 1911. New house.
20. Case 281 developed pellagra here in 1906.
21. Case 278 developed pellagra here in 1910.
22. Case 278 now living here.
23. Case 241 lived here in 1907.
24. Case 233 developed pellagra here in 1910.
25. Case 232 developed pellagra here in 1910.
26. Case 277 developed pellagra here in 1907.
27. Case 277 now living here.
28. Case 235 developed pellagra here in 1911. Died.
29. Case 211 died of pellagra here in 1911.

Mosquitoes are prevalent in this village and screened houses are the exception. A number of the pellagrins here stated to me that bed-bugs were present in their houses, but no specimens were collected. The water used in this village is obtained from deep-driven wells located at various

places. Much of the food is obtained at the store near the railroad switch; other stores are patronised.

It may be of interest to note here that Mr. W. V. KING, of the Bureau of Entomology, United States Department of Agriculture, stated to me that he had collected specimens of the larva of the fly *Simulium* from the waters of the little creek to the east of this village.

Still other communities, both in the coal-mining and in the cotton-mill sections, were visited, and were found very nearly similar to the above with reference to the pellagra situation. For various reasons they could not be worked up as thoroughly as the two districts given above. Further work similar in nature to these studies and with greater attention to detail is contemplated for the coming summer of 1912.

I am indebted to Passed Assistant-Surgeon C. H. LAVINDER for many helpful suggestions during the preparation of this article, and for his permission to present it for publication.

Series Case	Name	Address	Date	Diagnosis
Seen with		Note		
Reported by		Nativity	Occupation	
Age	Sex	Race	M S W	PELLAGROUS (Present Attack).
FAMILY				
		Date of Onset		
		Residence at	(Town)	(State)
		Number and Health of Family		
Children	Marriage			
		Food		
		Water Supply		
Remarks	Residences	City, Town, Rural		
		Relation to Water Courses		
		Remarks		

Fig. 1. Face of Card.

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PELLAGROUS (FIRST ATTACK).

Date of.....

Residence at.....

Number and Health of Family.....

Relation to Other Cases.....

To Heredity

To Animals.....

Food

Corn

Water Supply

To Water Courses

Remarks

.....

.....

.....

ENVIRONMENT AND CONDITIONS.

Topography

Location of House—City, Town, Rural

Economic

Sanitation

Water Courses.....

Water Supply.....

Remarks

.....

.....

.....

Fig. 2. Reverse of Card.

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